Volume 1

The Psychrometric Control of House Dust Mites: Testing the Validity in UK Dwellings of Two Combined Hygrothermal Population Models for Beds

Marcella Ucci

The Bartlett School of Graduate Studies

University College London

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ProQuest LLC
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106-1346

Student Declaration:

The author of this thesis, Miss Marcella Ucci, confirms that the work presented in this thesis is her own. Where information has been derived from other sources, the author confirms that this has been indicated in the thesis.

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Abstract

Beds are a crucial source of house dust mite (HDM) allergens, which play a major role in allergic disease, particularly asthma. HDM require a specific combination of hygrothermal conditions to thrive. These bed conditions depend on a number of interacting factors, such as: external climate; building characteristics; heating, ventilation and moisture-producing habits; mattress properties; etc. Because of the complexity of the many interacting factors occurring in real dwellings, a modelling approach is required, whereby the models' predictions have to be consistent with field results.

This thesis tested the hypothesis that a combined HDM population-hygrothermal model for beds can adequately predict field data and that the model can be a valuable tool for scenario modelling and intervention studies focused on the psychrometric control of house dust mites in UK housing. Two combined models were considered: a simple steady-state one-dimensional model (BED/MPI), and a complex transient three-dimensional model (Lectus/Popmite). A combination of fieldwork and scenarios modelling was carried out, which involved hygrothermal and mite monitoring of 25 beds, utilising a novel technique whereby live mites were caged in 'mite bags' and installed in monitored beds and bedrooms (82 sets of mites bags). The work was carried out as part of a multidisciplinary project aimed at developing and testing the models.

Good agreement was found between field data and the models' predictions, particularly when the uncertainties due to input variables and measurements were taken into account. The results showed that under borderline conditions for HDM growth, simple steady-state predictions may not be accurate. Temperature, not only RH, is a critical variable for HDMs. Areas for model improvement were also identified. In particular, factors other than hygrothermal conditions may be crucial for a bed's mite carrying capacity, requiring further investigation: food, space availability, and mite movement. Despite these uncertainties, it can be concluded that greater ventilation and reduced moisture rates can decrease mite levels in beds. The ventilation rates provided by some mechanical ventilation with heat recovery systems may be inadequate to sufficiently control moisture and reduce mite growth. Scenarios modelling suggests that there is considerable potential for the psychrometric control of house dust mites in UK dwellings.

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- HDM: House Dust Mites
- RH: Relative Humidity
- DP: Dermatophagoides Pteronyssinus
- DF: Dermatophagoides Farinae
- CEH: Critical Equilibrium Humidity
- VPX: Vapour Pressure Excess
- MPI: Mite Population Index
- MVHR: Mechanical Ventilation with Heat Recovery

CHAPTER 1:

INTRODUCTION

CHAPTER 1: INTRODUCTION

1.1 Background

House Dust Mites (HDMs) are commonly found in bedding and soft furnishings. Exposure to mite allergens (mostly present in their faecal pellets) can lead to sensitisation, and to exacerbation of asthma symptoms (National Academy of Sciences, 2000), as well as of other allergic conditions such as eczema and allergic rhinitis. Some authors warn of a current "epidemic" of allergy and asthma (Holgate, 2004; Eder *et al.*, 2006), based on the findings of several epidemiological studies which reported an increase in the occurrence of allergic diseases over the past 30-40 years - particularly in affluent countries (e.g. von Herzen and Haahtela, 2004). The UK has one of the highest rates of asthma symptoms in Europe (ISAAC Steering Committee, 1998), and a UK Report on 'Building Regulation, Health and Safety' ranked the health risk posed by HDMs in the highest-level group (Raw, 2001).

Mites require specific hygrothermal conditions for their survival, thriving in warm and humid environments. Studies have shown that in high altitudes – where the air is drier – mite numbers and asthma cases are low (Charpin, 1988). Theoretically, by controlling the hygrothermal conditions of the mite microclimates (psychrometric control), it is possible to reduce/eradicate mite numbers (Arlian *et al.*, 2001; Cunningham, 1996). Heating and ventilation have been used in a number of studies to control the hygrothermal conditions in housing, hence reducing mite infestation levels (e.g. Pretlove, 2002; Hirsch, 2000; Howieson, 2005).

Many strategies other than the psychrometric method are available for the control of HDM populations and/or allergens. These include: high-efficiency vacuums, steam cleaning, mite-proof barriers, acaricides, etc. However, most of these strategies can be time-consuming, while the psychrometric approach could be "built-into" housing design or refurbishment. This could potentially prevent sensitisation to HDM allergen to occur in the first place, and significantly save on the cost of controlling allergic conditions, where for example asthma has been reported as costing the UK £2 billion a year (Chaytor, 2003). Although it is still debated whether any of these mite control strategies can reduce mite infestation to

a level sufficient for health benefits (Gøtzsche, 2006), there is some consensus that a combination of strategies is probably the most effective method (Cunningham, 1996).

Although it is generally accepted that mites thrive in humid environments, there is some controversy about the feasible humidity threshold required to prevent mite infestation in UK housing (Lowe, 2000). This is also because most studies on hygrothermal conditions and mite survival have been conducted under steady-state conditions, with mites reared over many generations under ideal hygrothermal conditions and with a very controlled unnatural diet, rather than under transient conditions with 'wild' mites, reared over a smaller number of generations and kept under transient conditions with a more natural diet (Crowther, 2000). More research is necessary on the ability of 'wild' mites to survive under transient conditions.

A number of studies (including UK-based studies) concluded that beds are an important source of mite allergen exposure, since mite allergen concentrations are often higher in beds (Hirsch *et al.*, 1998; Simpson *et al.*, 2002). Hygrothermal conditions in beds are very variable, depending upon a number of interacting factors, such as: climate; building characteristics (especially insulation and air-tightness); heating and ventilation patterns; occupants' moisture production; type of mattress; length of time the mattress is occupied, etc. By establishing how such factors interact and affect mite survival, it should be possible to identify which building features and occupant behaviours should be adopted in order to prevent mite growth in beds.

1.2 Problem statement

The previous section highlighted that beds are a crucial source of mite allergens, which play a major role in allergic disease, particularly asthma. House dust mites require a specific combination of hygrothermal conditions to thrive. Since mite food (i.e. skin scales) is generally plentiful in beds, the main driver for dust mite infestation of a bed is its hygrothermal conditions, which are affected by bedroom conditions.

Some authors advocate that the rise in UK asthma levels may be due to recent changes in the building stock, where energy efficiency concerns may have caused excessively low ventilation rates in housing, resulting in high moisture levels which create favourable conditions for house dust mite infestations (Howieson *et al.*, 2003). However, it is extremely difficult to test this hypothesis and most existing data are inadequate for conclusions to be drawn whether ventilation rates *directly* cause ill-health (Davies *et el.*, 2004). Indeed, any study attempting to establish a link between health outcomes and changes in HDM levels (e.g. in beds) resulting from modifications in building design/use is faced with a large number of variables and mechanisms, some of which are difficult to measure or not fully understood. Figure 1.2.1 highlights the main variables which should be taken into account when considering the links between: the building stock, HDM levels in beds, and health outcomes – with a focus on the psychrometric control approach.

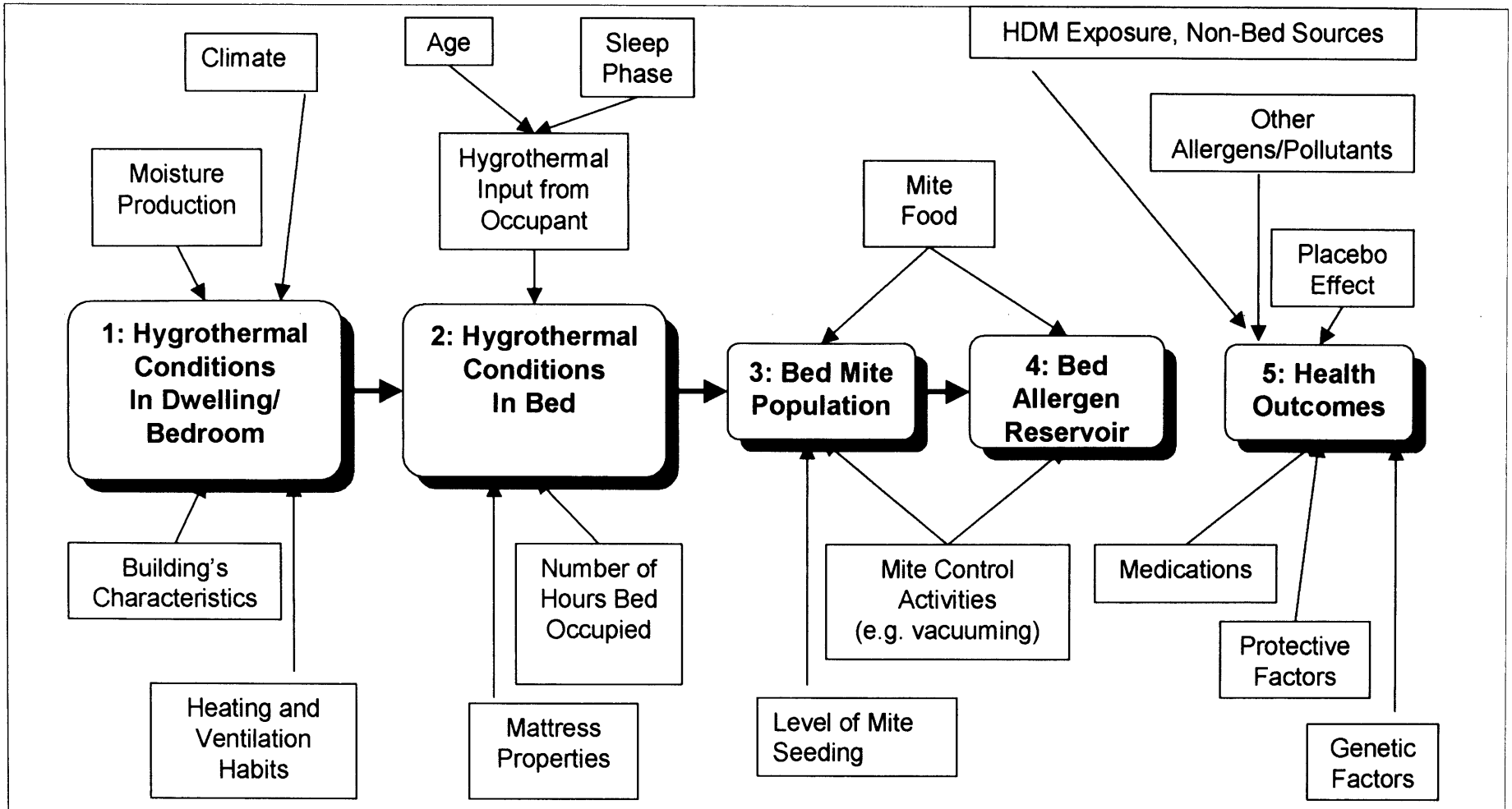

Fig 1.2.1 Postulated pathways between hygrothermal conditions in dwellings, HDM levels in beds and adverse health outcomes

Figure 1.2.1 shows the postulated pathways between hygrothermal conditions in dwellings and adverse health outcomes. The hygrothermal conditions in a bedroom have an impact on the conditions of its bed, which in turn affect the growth of a HDM population in such bed. This HDM population will produce a certain quantity of allergen (i.e. faeces), at a rate which is also dependent on the hygrothermal conditions to which the mites are exposed. The accumulated allergen might result in an adverse health outcome. The health outcome could be: 1) sensitisation to HDM allergen; 2) development of allergic disease (e.g. asthma); or 3) exacerbation of allergic disease symptoms. However, for every step of the postulated pathway, a number of variables are involved, which are illustrated in the graph. Ideally, all of these variables should be measured, in order to fully understand the links between building design/use, dust mite infestation in beds, and health outcomes. However, the measurement of all these variables is extremely expensive and in some cases it is rather difficult (e.g. ventilation rates, asthma). Furthermore, a multidisciplinary approach is required, involving: building physicists, acarologists, epidemiologists and respiratory health experts. However, it is often difficult to create links between such different disciplines, and to obtain funds for multidisciplinary studies. In addition, the postulated pathways illustrated in Figure 1.2.1 are based on a number of underlying mechanisms, some of which are still not fully understood (e.g. asthma development; impact of transient conditions on mites), and some may be complex, non-linear relationships (e.g. allergen exposure and sensitisation). Based on all these limitations, it is hardly surprising that no study could conclusively prove the clinical efficacy of the psychrometric HDM control method (Gøtzsche, 2006).

Given the complexities discussed above, it is helpful to break the problem into a number of more manageable research questions. In order to assess the potential efficacy of the psychrometric control method and associated mechanisms, two issues have to be considered: 1) How the building design/use affect hygrothermal conditions in beds, 2) How bed hygrothermal conditions affect mite growth. As illustrated in Figure 1.2.1, these issues alone are affected by a number of interrelated variables. For example, house dust mite growth is dependent on both temperature and RH, which are interrelated. In energy efficient dwellings, low ventilation rates might result in higher moisture concentrations, but higher

insulation levels should also increase indoor temperatures, producing a reduction in relative humidities. It is therefore important to establish as accurately as possible how building characteristics affect room conditions. On the other hand, a population of mites in a bed does not directly experience the hygrothermal conditions of the bedroom, since bed conditions are also affected by other factors, such as: the properties of the mattress, the length of time the bed is occupied, etc. Furthermore, different combinations of temperature and RH can affect mite populations in disparate manners. Because of the complexities of the many interacting factors, a modelling approach is required, untangling the mechanisms underlying the first three steps illustrated in Figure 1.2.1, which is the focus of this thesis. In this case a modelling approach requires in effect a combination of 3 models: 1) A building model, predicting how building characteristics, occupant behaviour and climate affect temperature and humidity in the bedroom; 2) A bed model, predicting the impact of bedroom hygrothermal conditions, mattress properties and occupant characteristics on bed hygrothermal conditions; 3) A population model, predicting the impact of bed hygrothermal conditions on mite populations. This combined model should enable the identification of those building features and occupant behaviours which can reduce mite infestations in beds.

As a result of a two-year research project[1] funded by EPSRC, a multidisciplinary UK-based team developed a "simple" and a "complex" combined model predicting the survival/decline in beds of *Dermatophagoides Pteronyssinus* (DP), the most common HDM species in the UK and Europe. The "simple" model is a steady-state one-dimensional model, which was developed so that it could be easily utilised by building designers or environmental health specialists, for the identification of "at risk" dwellings or design solutions. The model takes into account most of the parameters that are considered relevant for the psychrometric control of DP mites. As illustrated in Figure 1.2.2, the simple model is split into three modules: a building simulation model predicting monthly hygrothermal conditions in the bedroom - such as the model Condensation Targeter (Oreszczyn and Pretlove, 1999)[2]; the bed model BED (Pretlove *et al.*, 2005); and the

[1] GR/M93925/01, May 2000-May 2002
[2] The Condensation Targeter model was not developed as part of the EPSRC-funded project on dust mites

population model MPI (Crowther *et al.*, 2006). The final output of this simple combined model is a monthly MPI factor for the bed, indicating whether an existing mite population in a bed would be growing or declining, under the hygrothermal conditions predicted by the BED model.

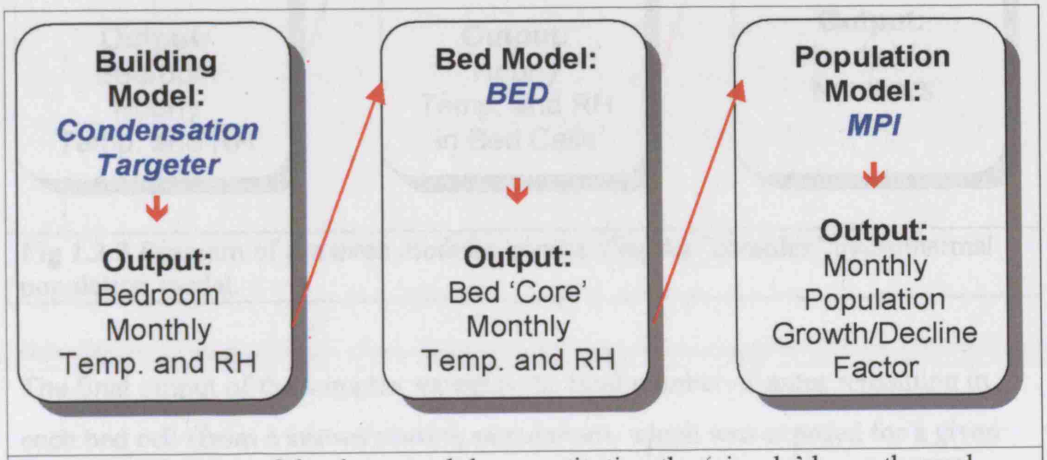

Fig 1.2.2 Diagram of the three modules constituting the 'simple' hygrothermal population model.

Although the simple model illustrated in Figure 1.2.2 is relatively easy to use, it is based on steady-state conditions. However, real dwellings experience variable hygrothermal conditions, depending on the season and on the time of the day. Furthermore, the simple model does not take into account the 3-dimensional heat and moisture flow in the bed. Therefore, the EPSRC-funded team also developed a three-dimensional transient complex model for the mattress and the mite population. As illustrated in Figure 1.2.3, the complex model comprises the bed model Lectus (Ridley *et al.*, submitted) and the population model Popmite (Biddulph *et al.*, 2007). The complex model also requires hourly bedroom conditions, which can be monitored values, or predictions from any commercially available software, such as EnergyPlus (US Department of Energy, 2007).

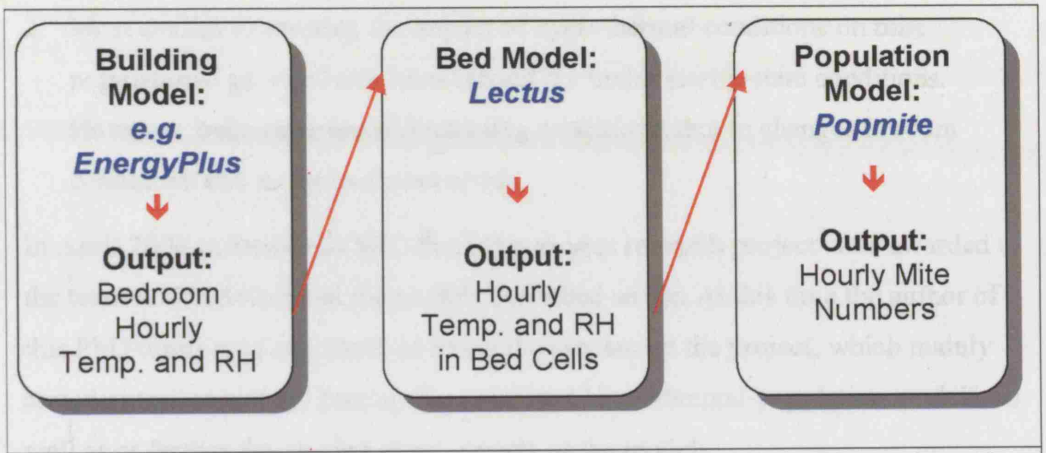

Fig 1.2.3 Diagram of the three modules constituting the 'complex' hygrothermal population model.

The final output of the complex model is the final number of mites remaining in each bed cell (from a known starting population), which was exposed for a given amount of time to transient hourly conditions. The complex model is aimed at expert-users for research purposes. A more detailed description of the models is provided in Chapter 3.

The simple and the complex models described so far were mostly developed on the basis of laboratory experiments and of first principles considerations. However, it is crucial that these models are tested in the field, for two main reasons:

1. The set-point for the thermoregulation of the human body is not constant, but fluctuates according to endogenous factors, such as: age, vigilance levels, sleep deprivation, thermal adaptation, fever, intake of food or fluids, posture during sleep, heat exposure before sleep, depression, etc (see Chapter 2). Therefore, the heat and moisture output from the human body into the bed varies *across* individuals as well as *within* individuals (e.g. food intake etc.). At present, it is unclear to what extent these factors affect the magnitude of changes in heat and moisture output from the human body into the bed. This is mostly because many studies on sleep where carried out in laboratory conditions, under controlled circumstances. Therefore, it is particularly important to test hygrothermal bed models in real settings – rather than simply in laboratory conditions.

2. Most studies examining the impact of hygrothermal conditions on mite populations' growth have been carried out under steady-state conditions. However, beds experience fluctuating conditions, due to changes in room conditions and to the bed's occupant.

In April 2004, a further EPSRC-funded two-year research project[3] was awarded to the team which developed the models described so far. At this time the author of this PhD thesis was appointed as research assistant on the project, which mainly aimed at testing and calibrating the combined hygrothermal-population models, as well as at further developing some aspects of the models.

The following section summarises the research hypothesis and the objectives of this thesis.

1.3 Research hypothesis and objectives

The previous section highlighted that a modelling approach is required, in order to enable the identification of those building features and occupant behaviours which should be adopted, in order to avoid mite growth in beds. The reduction of mite infestation levels in housing ultimately aims to decrease HDM allergen exposure, potentially preventing adverse health effects such as the exacerbation of allergic symptoms.

This thesis focuses on testing two existing combined hygrothermal population models[4], based on field data. In particular, this thesis aims to test the hypothesis that: "a combined HDM population-hygrothermal model for beds can adequately predict field data and that the model can be a valuable tool for scenario modelling and intervention studies focused on the psychrometric control of house dust mites in UK housing".

The objectives of this thesis are:

1. Establish whether the models' predictions are satisfactory, in relation to fieldwork data;

[3] GR/S70678/01, Apr. 2004-Apr. 2006
[4] Developed during the EPSRC project GR/M93925/01, in 2000-2002.

2. Assess the models' capabilities, including the advantages and limitations of the steady state model versus the transient model.
3. Ascertain the scope for using the models in order to establish adequate design and occupant behaviour strategies for the psychrometric control of house dust mites in UK dwellings.

These objectives were successfully accomplished through a combination of fieldwork and scenario modelling. It should be mentioned that the models examined in this thesis can be used for assessing the potential for the psychrometric control of HDM populations in any climate. However, this thesis mostly focuses on the UK, which is taken as a case study.

The field data analysed in this thesis was gathered during the previously mentioned EPSRC-funded project[5] - mostly by the thesis author - and it was collected with an aim of testing and further developing the 2 combined hygrothermal population models. The following section provides further details of the research project - including the role of the author, in relation to the originality of this thesis.

1.4 Originality and novelty

As illustrated in the previous section, this PhD was carried out as part of a EPSRC-funded 2 years research project (2002-04), where the author was employed as research assistant. The research project aimed to test and further develop two combined hygrothermal population models, based on laboratory and field studies. The project required a multidisciplinary approach, with expertise in building science, hygrothermal properties of materials, acarology and population modelling. Each expert did not work in isolation, since the various elements of the research project relied on each other. This section provides further details on the research project - including the role of the author, in relation to the originality of this thesis.

Some initial validation work had already been carried out in the initial model development project (2000-2002). However, the new research project (2004-

[5] GR/S70678/01, Apr. 2004-Apr. 2006

2006) aimed at testing both hygrothermal models against a lager number of beds and within realistic conditions. The author of this thesis carried out all the hygrothermal monitoring of bedrooms and beds for this purpose.

As previously mentioned, it is crucial to test the population model's prediction against the range of transient hygrothermal conditions commonly found in real beds. However, the realistic reproduction of fluctuating hygrothermal conditions can be difficult and expensive in a laboratory setting. On the other hand, it is not possible to assess the *direct* impact of hygrothermal conditions on existing mite populations in real beds, due to difficulties associated with sampling live mites – which for example cling to mattress fibres when an attempt is made to vacuum them. Consequently, the research team developed a new technique, which involves caging live DP wild mites and food in a mite and allergen proof 'bag' (similar in size to tea bags) made from porous material. The 'mite bags' are placed in a bed with monitored hygrothermal conditions, and after six weeks they are retrieved and the live mites counted. Although the excess food supply and the lack of freedom to move are unrealistic, this technique gives the opportunity to use real occupied beds as 'incubators' (in a way that is acceptable to an Ethical Committee), where mite growth can be examined in relation to real transient conditions, and compared to the population model's predictions (Popmite). The 'mite bags' were manufactured by Toby Wilkinson (KTP associate, Cambridge University and Medical Entomology Centre), who also counted the mites at the beginning and at the end of the monitoring period.

The EPSRC project also included laboratory work carried out by Dr Barbara Hart (Royal Agricultural College, Cirencester) on 'wild' DP mites - i.e. not reared under laboratory conditions. The work aimed to assess the 'wild' mite performance in relation to different (steady-state) hygrothermal conditions and different food types, in comparison with 'lab mites' (Hart B *et al.*, 2007). Furthermore, laboratory studies were carried out by Toby Wilkinson, to assess the effect of a range of temperature and RH combinations on 'wild' DP mites, as well as different lengths of time exposed to unfavourable conditions.

The results of the lab work were then used at the end of the research project to further develop the Popmite model, which was carried out by Dr Phillip Biddulph (previously Cambridge University, now UCL).

In summary, the 2004-06 EPSRC project included laboratory experimentation with 'wild' mites, and validation fieldwork on the hygrothermal conditions in beds and on the survival rates of caged mites in real beds. The fieldwork study was carried out by the author, in collaboration with Toby Wilkinson. In particular, Mr Wilkinson was responsible for producing and counting the mite bags, whilst the author was responsible for the hygrothermal monitoring of bedrooms and beds, as well as surveying the monitored dwellings/bedrooms and the study participants. However, it should also be highlighted that the author of this thesis was a core part of the research team, being one of two full-time researchers working on the project. Although the author played an active role in the project - which was equal to the other investigators in the team – this thesis does not focus on the *whole* research project, where it might be difficult to isolate the individual elements of originality, but rather on the specific tasks which were particular to the author of this thesis: i.e. testing the models against field data. However, the key advances of the project *as a whole* are here briefly summarised, in order to illustrate the breath of research activities in which the author was involved:

1 A new methodology has been established for carrying out experiments with wild (as opposed to laboratory-bred) mites. Diet was found to be a significant factor affecting mite performance, particularly in sub-optimal conditions.

2 An innovative method has been successfully developed and tested ('mite bags') for measuring how mite population growth is affected by hygrothermal conditions over time, of relevance to both field and laboratory studies.

3 A comprehensive and consistent data set has been collected that describes the physiology of wild *Dermatophagoides pteronyssinus* house dust mites in steady state conditions for 26 different temperature and RH combinations.

4 The population model has been revised to take account of the new data set and has been extended with new sub-models for water balance, egg production/development.

5 The hygrothermal model now incorporates improved data for the thermal properties of bedding materials.

6 The validity of the hygrothermal and population models has been tested against a unique data set arising from three field studies involving a total of 25 houses. The population model's ability to simulate the effect of fluctuating conditions has also been validated against new laboratory data.

7 The efficacy of using modelling techniques has been tested for determining the most appropriate ways of controlling mite populations by modifying hygrothermal conditions for volunteer households in a range of house types. This pilot study demonstrated the potential impact of our models in predicting how to improve the health of asthmatics and has provided valuable insights of considerable relevance to future research projects.

Relevant published papers are provided in Appendix A.0.

The novelty of this thesis is due to the fact that: 1) No population models other than Popmite are currently available, capable of simulating *fluctuating* hygrothermal conditions, nor based on *'wild'* mite data; 2) No models other than Lectus are currently available that simulate transient *three-dimensional* heat and moisture transfer in beds; 3) No other hygrothermal bed models have been tested against *several real beds*; 4) The 'mite bag' technique is unique and it allows testing the population model against hygrothermal data from real beds.

These advances would not have been possible, had a multidisciplinary team not worked together. This is the advantage of multidisciplinary research, whose great benefit is producing original research outcomes, which could not have been achieved if working in isolation. As a result, this PhD *is* original, firstly because of the uniqueness of its outcomes - which could not have been achieved without a certain degree of dependency on other disciplines/experts. Secondly, most of the data utilised for the analysis was gathered by the author – apart from the 'caged mites'. Thirdly, all the data-analysis and discussions presented in this thesis was carried out by the author. Lastly, the author played a full and active role in the development of the project, and for example was fully engaged in the unique development of the concept of the 'mite bags' as a novel method for monitoring mites under realistic conditions. Furthermore, the author played a pivotal role in

setting up the methodology and in carrying out the data collection and analysis for the pilot intervention study (Series 3 study, see Chapter 4).

House dust mites pose a high health risk for the UK population. This research will help establishing to what extent the models can be used in order to assess which building's features and occupant behaviour might be adopted, in order to reduce mite growth and associated health risks.

The following section describes the thesis structure.

1.5 Structure of the thesis

This section illustrates the structure of the thesis, chapter by chapter.

Chapter 1 briefly introduces the background to the dust mite problem, and identifies the problem statement and the objectives of the thesis, including its hypothesis.

Chapter 2 reviews the relevant published literature, including: the health effects of house dust mites, their preferred environmental conditions, sampling mite numbers and their allergen levels, HDM control methods, housing characteristics associated with mite infestation, issues associated with the hygrothermal modelling of beds.

Chapter 3 describes the models tested in this thesis.

Chapter 4 describes the methodology utilised to demonstrate the research hypothesis.

Chapter 5 examines the comparison between fieldwork results and Lectus predictions.

Chapter 6 examines the comparison between fieldwork results and BED predictions.

Chapter 7 examines the comparison between fieldwork results and Popmite predictions, and it also illustrates the mite bags results.

Chapter 8 examines the comparison between fieldwork results and MPI predictions.

Chapter 9 includes a sensitivity analysis of the models.

Chapter 10 compares the simple versus the complex models.

Chapter 11 discusses the applicability of the model(s), by describing their use in a pilot study.

Chapter 12 illustrates a number of scenarios modelling, including the impact on HDM populations of changes in heating and ventilation, and of geographical location within the UK.

Chapter 13 discusses the findings of the thesis, drawing its final conclusions and recommendations for future work.

The Appendix includes additional graphs and an additional section related to the literature review. Papers where the author of this thesis is included in the authors' list are also provided.

Chapter 1: References

Arlian LG, Neal JS, Morgan MS, Vyszenski-Moher DL, Rapp CM, Alexander AK, (2001), Reducing relative humidity is a practical way to control dust mites and their allergens in homes in temperate climates, *Journal of Allergy and Clinical Immunology*, 107 (1): 99-104.

Biddulph P, Crowther D, Leung B, Wilkinson T, Hart B, Oreszczyn T, Pretlove S, Ridley I, Ucci M, (2007), Predicting the population dynamics of the house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle, *Experimental and Applied Acarology*, 41(1-2): 61-86.

Charpin D, Kleisbauer JP, Lanteaume A, Razzouk H, Vervloet D, Toumi M, Faraj F, Charpin J, (1988), Asthma and allergy to house-dust mites in populations living in high altitudes, *Chest*, 93 (4): 758-61.

Chaytor Mr, (2003), Oral Answers to Questions, Commons Hansard Debates, text for Tuesday 6 May 2003, 404 (389), Column 666.

Crowther D, Horwood J, Baker N, Thomson D, Pretlove S, Ridley I, Oreszczyn T, (2000), *House Dust Mites and the Built Environment: a Literature Review,* http://www.ucl.ac.uk/bartlett-housedustmites/publications.htm. Website checked October 2006.

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

Cunningham MJ, (1996), Controlling Dust Mites Psychrometrically - a Review for Building Scientists and Engineers, *Indoor Air*, 6: 249 - 258.

Davies M, Ucci M, McCarthy M, Oreszczyn T, Ridley I, Mumovic D, Singh J, Pretlove S, (2004), A Review of Evidence Linking Ventilation Rates in Dwellings and respiratory Health – A Focus on House Dust Mites and Mould, *The International Journal of Ventilation*, 3(2): 155-168.

Eder W, Ege MJ, and von Mutius E, (2006), The asthma epidemic, *New England Journal of Medicine*, 355(21): 2226 – 2235.

Gøtzsche PC, Johansen HK, Schmidt LM, Burr ML, (2006), House dust mite control measures for asthma, *The Cochrane Library*, 3: 1-38.

Hart BJ, Crowther D, Wilkinson T, Biddulph P, Ucci M, Pretlove S, Ridley I, Oreszczyn T, (2007), Reproduction and development of laboratory and wild house dust mites (Acari: Pyroglyphidae) and their relationship to the natural dust ecosystem, *Journal of Medical Entomology,* 44(4): 568-574.

Holgate ST, (2004), The epidemic of asthma and allergy, *Journal of the Royal Society of Medicine*, 97(3): 103-10.

Hirsch T, Hering M, Burkner K, Hirsch D, Leupold W, Kerkmann ML, Kuhlisch E, Jatzwauk L, (2000), House-dust-mite allergen concentrations (Der f 1) and mold spores in apartment bedrooms before and after installation of insulated windows and central heating systems, *Allergy*, 55(1): 79-83.

Hirsch T, Range U, Walther KU, Hederer B, Lässig S, Frey G, Leupold W., (1998), Prevalence and determinants of house dust mite allergen in East German homes, *Clinical & Experimental Allergy*, 28: 956-964.

Howieson SG, Lawson A, McSharry C, Morris G, McKenzie E, Jackson J, (2003), Domestic ventilation rates, indoor humidity and dust mite allergens - Are our homes causing the asthma pandemic?, *Building Services Engineering, Research and Technology*, 24(3): 137-147.

Howieson SG, (2005), *Housing and asthma*, London: Taylor & Francis.

ISAAC Steering Committee, (1998), Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. The International Study of Asthma and Allergies in Childhood, *Lancet*, 351 (9111): 1225-32.

Lowe R, (2000), Psychrometric control of Dust mites in UK Housing, *Building Services Engineering Research and Technology,* 21 (4): 274-276.

National Academy of Sciences (Committee on the Assessment of Asthma and Indoor Air, Institute of Medicine, U.S.), (2000), *Clearing the Air,* Washington: National Academy Press.

Oreszczyn T and Pretlove S E C, (1999), Condensation Targeter II: Modelling surface relative humidity to predict mould growth in dwellings, *Building Services Engineering Research and Technology*, 20 (3) 143 – 153.

Pretlove SEC, Critchley R, Howard R, Oreszczyn T, (2002), The Nottingham energy, health and housing study: reducing relative humidity, dust mites and asthma, *Building Services Engineering Research and Technology*,23(1): 43-55.

Pretlove SEC, Oreszczyn T, Ridley I, Wilkinson T, Crowther D, (2005), A steady-state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements, *Building Services Engineering Research and Technology*, 26(4): 301-314.

Raw GJ, Aizlewood CE, Hamilton MR, (2001), *Building Regulation, Health and Safety,* Garston (UK): BRE.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (*submitted*), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air.*

Simpson A, Simpson B, Custovic A, Cain G, Craven M, Woodcock A, (2002), Household characteristics and mite allergen levels in Manchester, UK, *Clinical & Experimental Allergy*, 32(10): 1413-9.

US Department of Energy, (2007), *EnergyPlus Version 2*, www.eere.energy.gov/buildings/energyplus/, website accessed: May 2007.

von Hertzen LC, Haahtela T, (2004), Asthma and atopy - the price of affluence?, *Allergy*, 59(2):124-37.

CHAPTER 2:

LITERATURE REVIEW

CHAPTER 2: LITERATURE REVIEW

2.1 Introduction

This chapter reviews the current published knowledge on the issues related to this thesis. In particular, this thesis aims to test two hygrothermal population models which predict the effect of room conditions on dust mite infestations in beds. As already highlighted in the previous chapter, this is a complex multidisciplinary issue, which links a number of disciplines: acarology (i.e. dust mite biology), building science, hygrothermal modelling and population modelling. The ultimate goal of the models tested in this thesis is the identification of those building features and occupant behaviours which avoid adverse health outcomes, through the reduction of dust mite infestations in beds. Figure 1.2.1 in the previous chapter illustrated how the relationships between housing, occupant behaviour, mattress characteristics, mite infestation and health outcomes is extremely complex. The models tested in this thesis address 2 of the 5 "steps" in the postulated pathways illustrated in Figure 1.2.2, namely: modelling the impact of bedroom hygrothermal conditions on beds, and modelling the impact of bed hygrothermal conditions on mites. Therefore, this literature review mostly focuses on issues associated with these two research topics. However, some background information is also provided on the links between dust mites and adverse health outcomes- especially asthma, which is the most serious condition associated with dust mites. The section on health in this literature review aims to: 1) provide some background information on the importance of HDM research, in relation to the "allergic epidemic", and to the role of house dust mites in allergic diseases; 2) Highlight how there are still many unanswered research questions on the mechanisms underlying the development of HDM sensitisation, asthma and symptoms exacerbations. These questions play a vital role for any research – such as HDM research - ultimately aimed at reducing adverse health outcomes.

This chapter is divided into 4 sections (apart from this introduction):

1. **HDM biology and its interaction with the environment**: Different methods are available for HDM control. One of these methods focuses on the importance of hygrothermal conditions on mite physiology, and on the idea of manipulating mite microclimates, in order to reduce/eradicate mite

infestations (psychrometric control). **Section 2.2** discusses HDM biology, with a focus on the role of hygrothermal conditions. Sampling methods are examined, as well as cross-sectional studies on housing characteristics and HDM infestations. HDM control methods are also discussed, with a particular focus on the psychrometric control method - including a review of intervention studies based on this method.

2. **Hygrothermal and population modelling**: Mattresses are an important source of mite allergens. Several factors affect hygrothermal conditions in beds. Furthermore, hygrothermal conditions affect mite population growth in a complex manner. A modelling approach is required in order to establish how the various factors interact and affect mite survival in beds. **Section 2.3** discusses the issues associated with the modelling of hygrothermal conditions in beds. Heat and moisture transfer in porous materials is discussed, together with the issue of hygrothermal properties of porous materials– particularly textiles. The thermoregulation of the human body is also examined, with a focus on sleep. Testing and validation of models is discussed. A review of existing models is also provided.

3. **The role of house dust mites in the "allergy epidemic"**: The ultimate goal of house dust mite (HDM) research is the reduction/eradication of HDM infestations, because of their health impacts. **Section 2.4** provides background information on the role of house dust mites on health – particularly asthma - set within the context of the rise in the prevalence of asthma and allergies observed in westernised countries in the past decades.

4. A **summary** of the main issues is discussed in this Chapter (section 2.5).

It should be highlighted that the following literature review is rather extensive, since a range of diverse topics had to be covered due to the complex multidisciplinary nature of this subject and hence of this thesis.

2.2 House dust mites, preferred microenvironments and control measures

In this section, the biology of house dust mites is examined, with a particular focus on their dependency on certain hygrothermal conditions (section 2.2.1). Sampling methods for house dust mites and their allergens are illustrated in section 2.2.2, while section 2.2.3 discusses associations between HDM allergen levels and housing characteristics. Available methods for HDM control are briefly discussed in section 2.2.4, with a greater focus on the psychrometric control method (section 2.2.5). Finally, section 2.2.6 examines a number of intervention studies on HDM psychrometric control.

2.2.1 House dust mite biology

The term house dust mites (HDM) usually refers to the mite family *Pyroglyphidae*. There are other domestic mites from other mite families, for example storage mites, which can also cause sensitisation in exposed individuals. From the Pyroglyphidae family, 3 species are more often found in house dust: Dermatophagoides pteronyssinus (DP), D. farinae (DF), and Euroglyphus maynei (EM) (Platts-Mills, 2nd WHO, 1992). In general DF is more commonly found in North America and in other regions with prolonged dry weather whilst DP (Fig 2.2.1) is abundant in areas with constantly higher humidity, such as UK, Australia and New Zealand. In the UK, DP is usually the most abundant mite species, followed by EM (Crowther *et al.*, 2000). Table 2.2.1 illustrates the classification of the house dust mite species DP.

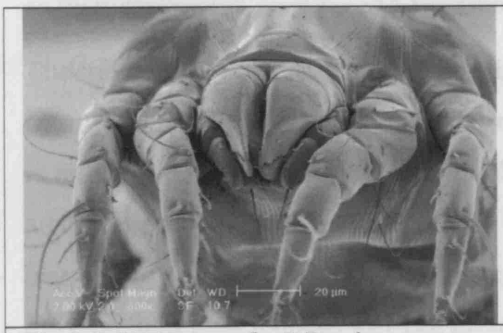

Fig 2.2.1 Picture of a DP mite (provided by: UCL)

Table 2.2.1 Classification of the HDM species *Dermatophagoides pteronyssinus* (Crowther *et al.*, 2000)

	Animalia
Phylum	Arthropoda
Class	Arachnida
Order	Acari
Suborder	Astigmata
Family	Pyroglyphidae
Genus	Dermatophagoides
Species	pteronyssinus

Mite *allergens* are divided into specific groups on the basis of their biochemical composition, sequence homology, and molecular weight. Two major groups of allergens from the genus Dermatophagoides have been identified, referred to as: group I allergens (Der p1 for DP, Der f1 for DF and Der m1 for DM) and group II allergens (Der p2 for DP, Der f2 for DF). Both allergens are mite digestive enzymes excreted in the mite faeces. Other sources of mite allergens include their body parts, including mite saliva that is left in the environment when mites feed (Arlian and Platts-Mills, 2001b). Mite faecal pellets are relatively large (diameter: 10-40 µm), which means they remain airborne for a relatively short time. Mite faecal particles of 20 µm in diameter usually carry 0.1-0.2 ng of mite allergen, while for example particles of 1 µm in diameter - cat particles or nebulised droplets - each carry only 10^{-4} to 10^{-6} ng of allergen (Platts-Mills *et al*, 1992). Most mite allergic individuals produce IgE antibody to both group I and group II allergens. Mite allergens are usually quantified in extracts of house dust by using enzyme immunoassays. Group I allergens are often used in studies, since the reagents for group I allergens are more widely available and the results from previous studies are easier to compare (Platts-Mills and de Weck, 1989).

Dust mites are approximately 0.3 mm in size and are normally invisible to the naked eye because they are translucent. They feed on human skin scales, as well as on other components of house dust. Consequently, HDM are more commonly found in those domestic environments were food is plentiful, such as mattresses and carpets, as well as soft toys and soft furnishings. Mites are photophobic and

this may be one of the reasons why they live within the depths of habitats, as well as their surfaces (Crowther and Wilkinson, *accepted for publication*).

The life cycle of house dust mites consists of 5 stages: egg, larva, protonymph, tritonymph, and adult. The duration of each life cycle stage and population growth is influenced by both ambient Relative Humidity (RH) and temperature. Egg-to-adult development time is significantly longer below 23°C in DP mites, thus hampering population growth even when the RH is high. As temperature rises, development time reduces and egg production also increases. Beyond a certain temperature, however, adult female longevity and egg production fall off sharply (Crowther *et al.*, 2006). Development from egg to adult requires 23 to 30 days for both *D. pteronyssinus* and *D. farinae* under optimum hygrothermal conditions (22 to 26 °C, 75% RH). The time required to complete the life cycle is lengthened or shortened with respectively lower and higher temperatures (Arlian, 1989).

House dust mites are 70-75% water by weight and they lose most water during egg production, by evaporation from the body, with body secretions and through defecation. HDM obtain little water through the usual routes of drinking water or through moist food. Their primary source of moisture is water vapour that is actively extracted from unsaturated air. This active mechanism involves secreting a hyperosmotic solution from the supracoxal glands that open just above their first pair of legs. The hygroscopic secretion absorbs moisture from the air whilst running along a small channel to the preoral cavity, where it is ingested. If the RH of the air is too low, the hygroscopic solution crystallizes and the mite starts to dehydrate, eventually dying. This critical low RH is often referred to as the *Critical Equilibrium Humidity* (CEH), defined as the RH below which mites are unable to maintain their water balance and lose water more rapidly than they gain it (Crowther and Wilkinson, *accepted for publication*). The further RH falls below CEH, the faster mites dehydrate and ultimately die, although the time required varies according to species and life cycle stage. The CEH for fasting DF mites ranges between 55 and 75% and is proportional with temperature. In DP mites, CEH is 73% at 25 °C but the dependency of CEH from temperature has not been proven yet, although it is reasonable to assume a similar temperature-dependence of CEH. Feeding mites may have a slightly lower CEH than fasting mites (Arlian, 1989). Furthermore, CEH depends on the mites' state of hydration (Crowther *et*

al., 2006). Therefore, it has been hypothesised that there is a *range* of values of CEH for any given temperature (Crowther *et al*, 2006).

High mould densities are likely to have a negative effect on mite populations, as a result of competition for space and deterioration of the food, as well as contamination by mould toxins. Since mould activity increases rapidly with rising RH, mite populations decline sharply above a certain RH level - approximately >80% for DP (Crowther *et al.*, 2006).

Mites have developed a number of strategies to prevent desiccation and increase their survival. When the ambient RH is below CEH, protonymphs may become quiescent with a significantly reduced metabolism compared to active protonymphs and with extreme resistance to desiccation. Therefore, quiescent protonymphs are likely to survive dry spells and form the source of breeding mites when RH is again optimal (Arlian, 1992). Furthermore, clustering of house dust mites has been observed and it was established that in *D. farinae* this adaptive behaviour helps reducing water losses in mite populations (Glass *et al.*, 1998). However, clustering does not appear to occur in *D. pteronyssinus* (Crowther and Wilkinson, *accepted for publication*).

The importance of hygrothermal conditions on mite populations is confirmed by the seasonal nature of dust mite allergen levels. Several studies found seasonal variations in mite allergen concentrations, which are associated with changes in population size due to variations in weather conditions. In temperate climates, mite allergen levels and mite numbers have been found to be higher in late summer and early autumn. A study found that Der p1 levels in winter, spring and summer were approximately 0.8 times the autumn levels, while for Der f1, the correction factors were 0.8, 0.6 and 1, for winter, spring and summer respectively, compared to autumn (van Strien *et al.*, 2002). However, within the same climatic area, large variations in mite infestation levels can exist across the housing stock, due to diverse hygrothermal conditions caused by different building characteristics and occupant behaviours (Crowther *et al.*, 2006).

Before discussing the methods currently available for HDM control (section 2.2.4), the next two sections discuss the methods for detecting mites and their

allergens, and those housing characteristics, which have been found to be associated with higher HDM infestation levels.

2.2.2 Mite sampling methods

Dust samples are the most commonly used method for assessing mite infestation levels, whereby the sampled dust is examined for mite numbers and/or mite allergens. The dust samples are usually collected with a vacuum cleaner, either by using a purpose-designed dust collector, or by using a clean dust-bag for each sample. Once the dust is collected, house dust mites can be removed from the dust using a flotation or suspension technique. This generally involves placing the dust sample in a saturated salt solution (NaCl), and collecting the floating mites for examination under the microscope (Crowther and Wilkinson, *accepted for publication*). This technique allows the identification of the predominant mite species, and the recognition of live, dead, larval or adult types. This procedure – which does not identify faecal particles or HDM allergens - requires a skilled acarologist and can be time consuming.

Mite allergens are usually quantified in extracts of house dust by using immunochemical assays. The most common technique is the enzyme-linked immunosorbent assay (ELISA), which requires a trained technician and sophisticated laboratory equipment. Other cheaper and less sophisticated techniques are available for assessing the level of HDM infestation. For example, guanine is an excretion product of arachnids; among arachnids, mites are much more abundant than spiders. Therefore, determination of guanine in house dust can give an indication of mite infestation levels. However, this technique cannot distinguish between mite species and it can be unreliable at times (Platts-Mills and de Weck, 1989).

The number of mites or the quantity of allergens collected in each dust sample is affected by several factors, such as: the length of time the object is vacuumed, the power of the vacuum cleaner; the surface area which has been sampled. In order to make the results more comparable, a standard vacuuming protocol has been suggested, where 1 m^2 is vacuumed for 2 minutes (Platts-Mills and de Weck, 1989). Usually bed linen is removed, but any mattress cover which was in place

for more than 3 month is left in place (Zock et al., 2006). Once the number of mites or the weight of allergen in a dust sample has been identified, mite (or allergen) levels are usually expressed by unit weight of sieved dust (mite or allergen *concentration*), or by unit of vacuumed area (mite or allergen *load*[2]). Expressing the results in terms of concentrations or loads has its advantages and disadvantages (Colloff, 1991). The main advantage of expressing the results in terms of mite/allergen *concentration* is that the results are more easily comparable with other studies, since they are not affected, for example, by the power of the vacuum cleaner. Furthermore, it can be difficult to estimate the surface area of certain objects such as soft toys. However, mite/allergen concentrations can be misleading for a number of reasons. Firstly, allergen concentrations may not be correlated with the total quantity of HDM allergens found in an object: i.e. an object might have a high allergen *concentration,* but a low overall *quantity* of allergen. Secondly, dust densities can vary for various substrates (e.g. pillows vs. carpets), and although the dust from a sample is usually sieved before analysis, some misleading results can still occur. Finally, concentrations should not be used in repeated sampling when dust pool has been added or depleted, such as in clinical trials of some mite control strategies (Colloff, 1991). Expressing the results in terms of mite/allergen loads overcomes most disadvantages of referring to concentrations, but it also have some disadvantages. For example, the amount of dust (and consequently of mites or allergen) collected by vacuuming 1 m^2 is affected by the power of the vacuum cleaner. The amount of dust and allergen is also affected by the design of the dust collector, which makes the results difficult to compare with other studies (Wickens *et al.*, 2004). However, it should be pointed out that a number of studies on HDM exposure levels and health outcomes found that their results on exposure/outcome did not change whether allergen levels were expressed in terms of concentrations or loads (e.g. van Strien *et al.*, 2002; Schram-Bijkerk *et al.*, 2006). Furthermore, some studies found a correlation between allergen concentrations and allergen loads (Simpson *et al.*, 2002: Pearson's coefficient: ~0.9, $P<0.001$; Tovey *et al.*, 2003: Pearson's coefficient: 0.39, $P=0.04$).

[2] The terms allergen load, allergen density, allergen mass or other terms are used by different authors to refer to Der p levels expressed in terms of $\mu g/m^2$. In this thesis, the term 'allergen load' is used to refer to μg of Der $p1/m^2$ of vacuumed area.

The assessment of mite infestation levels via the analysis of dust samples has a number of limitations. Firstly, mites have sucker-like pulvilli at the ends of their legs, which allow them to cling firmly to their substrate. Consequently, vacuuming only remove a fraction of live mites, which has been estimated as little as 10% of the total population (Crowther *et al.*, 2000). Furthermore, mites live not only on the surfaces of their habitat but also deep within it. Consequently, vacuuming a mattress, for example, may only give a representative picture of the mite infestation levels of its top layer. Heat extraction is sometimes used to overcome these limitations. Heat extraction involves the application of heat on the back of a textile object. The mites move away from the heat and get trapped in the adhesive membrane applied to the other side of the textile. However, this technique has its limitations, the main one being that it can only be used when heat can be applied to one side of an object (Hill, 1998). It should also be mentioned that despite standardisation, differences in vacuum cleaner and dust collectors may also affect the sampling results, particularly for allergen loads. Therefore, mite sampling is far from an exact science.

Mite allergens can be asymmetrically distributed in domestic dust reservoirs. Howieson (2005) highlights that the habitats of living mites, the site of the initial faecal pellet deposition and the re-distribution of allergens - which may have been temporarily suspended – are not necessarily coincident. Howieson hypothesises that for example draughts or periodic air currents driven by convection may deposit airborne particles in specific niches. Mitakakis *et al.* (2002) compared different vacuuming procedures and concluded that sampling 4 *non adjacent* 0.25 m^2 areas that are not equidistant from the door is recommended when reservoirs concentrations of Der p1 are being measured.

HDM allergen levels found in dust are not necessarily representative of exposure levels, which require the allergens to become airborne. Air sampling of HDM allergens is not very common, mostly for the cost and practicalities involved. Furthermore, HDM faecal pellets are 10-40 μm in diameter and settle rather quickly from the air, although smaller particles of Der p1 may exist in very low concentrations. Therefore, air sampling may be useful only during periods of domestic activity such as house cleaning (Luczynska, 1998a). Tovey *et al.* (2003) compared 4 sampling methods for Der p1. Airborne allergen was sampled by a

Institute of Occupational Medicine static sampler. Also, settling dust was collected on Petri dishes, as well as via an adhesive-membrane system (A-book). Furthermore, vacuumed reservoir dust samples were collected from floors at the end of 1 week, and expressed both in terms of concentrations and loads. The authors concluded that no method can be considered ideal and recommended the use of both vacuum cleaner and Petri dishes. A more recent study on intranasal air sampling concluded that nasal air samplers confer no advantage over reservoir dust analysis for studies of asthma severity (Gore *et al.*, 2006).

This section reviewed the most commonly used methods for sampling mites and their allergens. Analysis of samples from vacuumed dust currently remains the most used and reliable method for the determination of mite infestation levels. The results obtained through this procedure should be expressed in terms of concentrations (per unit weight of sampled dust), as well as of loads (per unit area of vacuumed area). HDM allergen analysis of settled dust in Petri dishes may add some additional information on HDM allergen exposure profile.

The next section provides an overview of a number of studies which have attempted to establish which housing and habitat characteristics correlate with mite infestation levels.

2.2.3 Mite infestation levels, mite habitats and housing characteristics

In the previous sections it was highlighted that climatic conditions are an important determinant of mite infestation levels, because of the role played by temperature and relative humidity in HDM physiology. However, within regions with similar climatic conditions, large variations in mite infestation levels have been observed amongst dwellings (Crowther and Wilkinson, *accepted for publication*). Furthermore, some studies have found no correlation between mite allergen levels in different habitats (i.e. floors, mattresses, pillows etc.) within dwellings (Couper *et al.*, 1998). Although indoor hygrothermal conditions are driven by climatic characteristics, they are also affected by building characteristics, occupant habits and by the specific characteristics of mite habitats (i.e. mattress, carpet, etc.). Furthermore, mite infestation levels are likely to be determined by other factors than those affecting – directly or indirectly -

hygrothermal conditions in dwellings. For example, there is some evidence that washing bedding at temperatures above 40 °C is associated with reduced Der p1 concentrations in the bedroom floor dust (Luczynska *et al.*, 1998). Determining associations between mite infestation levels and housing (or habitat) characteristics can be an important way of understanding which features should be modified in a dwelling, in order to reduce mite infestation levels. Indeed, a study concluded that housing characteristics not only have a major impact on mite allergens, but also on respiratory morbidity and sensitisation independently, suggesting worsening of symptoms as well as a causative relationship with disease development (Hesselmar *et al.*, 2005). Consequently, a number of studies – mostly cross-sectional – have attempted to establish which housing and habitat characteristics correlate with mite infestation levels. Most of these studies have focused on HDM allergen rather than mite levels, mostly because allergens rather than mite themselves are directly responsible for adverse health outcomes. The results of these studies are reviewed in this section.

Since temperature and RH affect mite population growth, several studies have attempted to correlate hygrothermal conditions found in dwellings with mite allergen levels. Although some studies did not find a significant correlation (e.g. Chan-Yeung *et al.*, 1995), most studies on hygrothermal conditions and mite infestations only used spot measurements of room temperature and RH, which are not necessarily representative of the conditions experienced by mite populations throughout the year. Furthermore, live mites are more directly affected by hygrothermal conditions, while a reservoir effects occurs for mite allergens which accumulate over time. In addition, spot temperatures are confounded by external climatic conditions. However, some studies have found a correlation between mite infestations and indoor humidity, as well as with measured air exchange rates (Harving *et al.*, 1993; Sundell *et al.*, 1995). In the Netherlands, a study on 175 asthmatics also found that greater Der p1 loads (μ/m^2) were associated with RHs > 50% ($P<0.01$) (van den Bemt *et al.*, 2006). However, these studies did not focus on a random population sample and were all based in regions with cold winter climates.

Since measuring hygrothermal conditions (and/or ventilation rates) can be expensive in large scale studies, many authors have focused on specific housing

factors which affect indoor hygrothermal conditions (such as natural vs. mechanical ventilation) and/or are representative of hygrothermal conditions (e.g. mould presence). Focusing on these factors also gives the potential advantage of being able to identify those housing characteristics which should be removed or included in dwellings, in order to reduce mite infestation levels. For example, a UK study found that an extractor fan in the kitchen was associated with lower Der p1 concentrations in both living room and bedroom floors. Also, having an open fireplace in the living room decreased Der p1 concentrations in the living room floor by approximately 85%. However, the presence of an extractor fan or of an open fireplace was not correlated with Der p1 concentrations in mattresses (Luczynska *et al.*, 1998). The role of an extractor fan on lower mite levels was also confirmed by a more recent international ECRHS study (Zock *et al.*, 2006). Ground floor construction (particularly concrete floors in direct contact with the ground) and floor level (basement/ground floor vs. upstairs) also appear to affect mite infestations, since the lack of insulation and the moisture from the ground can be crucial for HDM growth, particularly in carpets (Crowther and Wilkinson, *accepted for publication*). In New Zealand, floor insulation was found the most important factor responsible for lower Der p1 levels in carpeted dwellings. Homes that did not have insulation fitted but had a room or a garage below the living room, or a thick layer of underlay below the carpet, also had lower Der p1 levels in at least one sample type (Wickens *et al.*, 2001).

In some studies, correlations have been found between mite allergens and factors which can be considered as proxies for specific hygrothermal conditions. For example, several studies found a positive association between mite allergen levels and: presence of mould, condensation, or damp, and number of people in the dwelling (Chan-Yeung *et al.*, 1995; Couper et al., 1998; Luczynska *et al.*, 1998; Dharmage *et al.*, 1999; Simpson *et al.*, 2002; Zock *et al.*, 2006). These factors are all indicators of high indoor moisture levels. Central heating was found to be associated with higher levels of mite allergens in two Australian studies (Dharmage *et al.*, 1999; Matheson *et al.*, 2003) but with lower levels in a UK study (Simpson *et al.*, 2002). Although warmer temperatures lead to lower RHs, lower temperatures also reduce mite development times. Furthermore, the presence of central heating might deter people from ventilating adequately their

dwellings, in order not to dissipate heat. The impact of air conditioning on mite allergen levels is also controversial, with an inverse correlation found in the US (van Strien *et al.*, 2004), but no correlation found in Australia (Chan-Yeung *et al.*, 1995).

Some studies found correlations between mite allergen levels and factors which are not solely linked to hygrothermal conditions. For example, the age of the mattress or of the carpet has been found to be positively associated with mite allergen levels, because of a reservoir effect (Luczynska *et al.*, 1998; Dharmage *et al.*, 1999; Simpson *et al.*, 2002; Zock *et a*l., 2006). A fitted carpet has also been found to be associated with higher HDM allergen levels in comparison with a smooth floor (Dharmage *et al.*, 1999; Simpson *et al.*, 2002), particularly for wool carpets (Dharmage *et al.*, 1999; Wickens *et al.*, 2001) - presumably because wool retains more moisture than other materials. The pile depth in the carpet and the carpet type (tufted or woven) also appear to affect mite allergen levels (Wickens *et al.*, 2001). For mattresses and pillows, the cover type appears to affect HDM allergen levels, probably because some covers act as a mite barrier. In mattresses, a cotton upper layer has been found to be associated with lower Der p1 concentrations as opposed to a synthetic upper layer. Mattress type (sprung, foam etc.) was found to affect HDM allergen levels in some studies but not in others, with some indication that sprung mattresses might have higher allergen levels than foam mattresses. This could be due to a combination of hygrothermal conditions determined by mattress properties, quantity of dust accumulated, availability of space, accessibility, etc. (van den Bemt *et al.*, 2006).

Most cross-sectional studies on the factors affecting HDM allergen levels have utilised statistical models in order to identify those variables which statistically explain the variability in HDM allergens. However, some authors point out that their models only explained 10-30% of the variance in allergen levels observed in their studies (Matheson *et al.*, 2003; van Strien *et al.*, 2004). This may be due to incorrect or insufficient variables examined in the studies, and/or to factors other than housing or habitats characteristics. It is also important to point out that most of the findings observed in these cross-sectional studies are true for a specific study population and regional area, and therefore should be applied to other contexts with caution. For example, a study carried out in a region/population

with very little use of carpeted floors would not be able to detect whether this floor type is associated with higher HDM levels, as found in other studies. Also, older dwellings have been found to be associated with higher levels of mite allergens in some studies (Australia: Dharmage *et al.*, 1999; New Zealand: Wickens, 2001; UK: Simpson *et al.*, 2002). However, in these studies the definition of 'older dwellings' may differ and the *characteristics* of an 'older dwelling' might change from one region to another. Zock *et al.* (2006) reported the results of a large multicenter Europe-based study on housing characteristics and allergen levels. Since some housing characteristics differ in various European countries, the results from Zock *et al.* might be useful in identifying the *main* determinants of HDM allergen in Europe. The study examined 3580 homes of participants to the European Community Respiratory Health Survey (ECRHS) from 22 study centres in Europe. In each home, mattress dust was analysed for Der p1, Der f1 and Der 2 allergens. The study found that low winter temperatures reduce Der p1 but not Der f1 levels. Important risk factors for high allergen levels included an older mattress, a lower floor level of the bedroom, an older building, and dampness (for Der p1 only). Sleeping with the window open in winter, the presence of an air brick or ventilation aperture in the bedroom, and of an extractor fan in the kitchen, were all associated with lower allergen levels in the mattress.

Since the findings from cross-sectional studies on mite infestations are specific to the examined population/region, the findings from 2 UK-based studies are reported here in more detail. Luczynska *et al.* (1998) examined the home environment of 158 adults aged 20-44 in Norwich, UK. Dust samples were collected in the living room floor, bedroom floor and mattress, and the dust samples assayed to determine Der p1 concentrations. The authors found that approximately 25% of living room floor and mattress, and over 30% of bedroom floor samples had Der p1 concentrations greater than 10 μg/g. Household characteristics associated with Der p 1 concentrations in both living room and bedroom were: floor level, extractor fan in the kitchen, and age of carpet. Living room Der p 1 concentrations were associated with: gas oven/hob, window condensation, open fires, vacuum cleaner type, smokers in the house and age of house. Bedroom Der p 1 concentrations were associated with: use of blankets and wash temperature of bedding. Mattress Der p 1 concentrations were associated

with: window condensation, concrete bedroom floor and age of mattress. Therefore, the authors highlighted that different household characteristics were associated with high Der p 1 concentrations in different parts of the house.

In a Manchester-based study, Simpson *et al.* (2002) recruited 564 adults, whose homes were representative of housing types in the area. Der p1 concentrations were assessed from dust in the living room floor, sofa, bedroom floor and mattress. The study found that Der p1 concentrations were highest in the mattress, and that two-thirds of homes contained Der p1 concentrations > 2 in at least one dust reservoir, and 40.3% contained Der p1 concentrations > 10 μg/g. There was a large range in Der p 1 levels between homes (> 10^{-3}-fold). Like in Luczynska *et al.* (1998), Simpson *et al.* found that certain factors affected some reservoirs more than others. Factors associated with higher Der p 1 levels in more than one dust reservoir were: older homes[3], older living room carpets, damp, condensation and mixed glazing. Age of the mattress was the strongest determinant of allergen levels in the mattress. There was also some evidence that the other factors associated with higher HDM allergen levels in the mattress were: absence of central heating, using electricity for heating, increasing age of the house, older living room carpet, damp in the bathroom, greater number of children in the house, damp. Twenty-four homes contained no detectable mite allergen, six of which reported damp. The authors concluded that mite allergen levels are high enough in two of every three homes to be associated with an increase in the risk of sensitization to mite. Although associations were found between housing characteristics and mite allergen levels, the latter were occasionally unpredictably very low in homes with risk factors for high levels.

Table 2.2.2 shows a comparison of different Der p1 concentration levels found in 4 UK studies, based on general population samples.

[3] In the study house age was divided into 4 categories: Before 1940; 1940-1960; 1960-1980; since 1980. Consequently, the study may not have been able to detect any impact on HDM allergens of more recent energy-efficiency measures (e.g. airtightness), if such impact exists.

Table 2.2.2 Der p1 concentrations in four UK-based studies

Geometric Mean (μg/g):	**Living Room Floor**	**Bedroom Floor**	**Mattress**
Study 1: Norwich	1.9	1.7	2.0
Study 2: Manchester	0.77	0.7	1.19
Study 3: Ipswich	(-)	(-)	0.77
Study 3: Norwich	(-)	(-)	1.30
Study 4: Ashford	1.5	(-)	(-)
Range (μg/g):	**Living Room Floor**	**Bedroom Floor**	**Mattress**
Study 1: Norwich	<0.1-259.0	<0.1-357.7	<0.1-819.8
Study 2: Manchester	0.05-215	0.05-310	0.05-1050
Study 3: Ipswich and Norwich	(-)	(-)	(-)
Study 4: Ashford	(1.26-1.85)*	(-)	(-)
Study Details			
Study 1: Norwich, 158 homes of adults aged 20-44 (Luczynska et al., 1998).			
Study 2: Manchester, 564 homes of adults (Simpson et al., 2002).			
Study 3: Two centres. 105 homes in Ipswich; 167 in Norwich. Adults aged 20-44 (Zock et al., 2006)			
Study 4: Ashford (Kent), 381 homes of children aged 4 (Torrent et al, 2006).			
(-) Data not provided. * 95% CI (range not provided).			

Finally, it is worth pointing out that higher HDM allergen concentrations have been found in mattresses (as compared to other habits) in different studies (e.g. Hirsch *et al.*, 1998), including a British study (Simpson *et al.*, 2002). However, mattresses may not always be the dominant HDM habitat, particularly when considering different mite species. Furthermore, mite levels might be affected by the depth at which they are sampled, since there is some indication that DF mites might crawl on top of the substrate more often than DP mites (Crowther and Wilkinson, *accepted for publication*).

This section reviewed a number of cross-sectional studies on mite allergen levels and housing (or habitat) characteristics. The main aim of these studies was to identify those characteristics that should be modified in dwellings, in order to reduce their mite allergen levels. The results from these studies are in some cases useful, but in most cases they cannot be generalised and/or applied to different contexts. There is clear evidence that the age of the sampled object (i.e. mattress, floor, etc.) is a determinant of HDM allergen levels, which is not surprising since HDM allergens are very stable. The impact of features which affect hygrothermal conditions (e.g. extractor fan, age of the dwelling, etc) is also clear in many studies. However, it is at times difficult to pinpoint which of these features is the most important. This is mostly because such features can affect hygrothermal conditions in different ways, depending from the housing stock, climatic

conditions, occupant characteristics, etc. There is however some evidence that in the European housing stock – including the UK – adequate ventilation (for example in the form of an extract fan in the kitchen) has the potential of reducing HDM allergen levels. Nonetheless, it is evident that many of the studies described in this section lack a multidisciplinary approach, having been designed either by epidemiologist with no clear understanding of building science, or by building scientists with limited knowledge of HDM biology or of statistical methods. Consequently, although the results of these studies are useful to give a preliminary idea on mite infestation and housing, they are far from conclusive. A multidisciplinary approach and a greater number of measured variables should yield better results. A greater understanding of how building features affect indoor hygrothermal conditions and of how these in turn affect mites - possibly with the aid of appropriate models - is necessary. The next sections discuss HDM control methods, including a number of intervention studies on the psychrometric control of house dust mites.

2.2.4 Methods for house dust mite control

A number of control measures can be utilised to control house dust mites. Detailed description and assessment of each method exceeds the scope of this thesis. This section is a brief overview of existing HDM control methods, including an introduction to the psychrometric control of house dust mites, which is discussed further in the next section (2.2.5).

It is important to distinguish between those HDM control measures aimed at preventing/reducing HDM infestations, and those aimed at controlling/removing HDM allergens which are very persistent in the environment. HDM control measures can be broadly divided into chemical, physical and hygrothermal methods, or any combination of these three methods. A WHO report on urban pests and health contains a chapter on house dust mites, which includes an extensive overview of these methods. Most of this section is based upon the information provided in the WHO report (Crowther and Wilkinson, *accepted for publication*).

Acaricides (e.g. benzyl benzoate or permethrin) are a chemical method which – if applied correctly – can kill mites, although it rarely reduces their allergens. The main disadvantage of acaricides is that they are meant to be used in settings where atopic individuals are likely to be found. Atopic subjects are generally susceptible to being sensitised to several allergens, and therefore exposure of atopic individuals to chemical agents may induce further sensitisation and/or exacerbation of allergic reactions.

Vacuuming is a physical method which is mostly aimed at removing HDM allergens, since live mites can cling very tightly to their substrate. However, regular vacuuming should also remove a source of food for live mites, at least from the top surface of their habitat. Vacuum cleaners with high efficiency filters are often recommended to asthmatics, since airborne allergen usually increases immediately after vacuuming with a standard vacuum cleaner (i.e. without HEPA filter).

Another physical method for HDM control is barrier fabrics, whose pore size is sufficiently small to block any passage of mites and their allergen, but adequate to allow moisture flow and therefore prevent sweating. Consequently, mite-proof fabrics can be used to encase pillows, duvets and mattresses in order to prevent mite colonisation and escaping of mite faecal matter.

A number of HDM control methods are also available, which utilise temperature and/or humidity control in order to reduce/kill house dust mites. Most of these hygrothermal methods do not remove mite allergens and are therefore often coupled with other methods such as vacuum-cleaning. High temperatures can be

used to kill mites. For example, Asthma UK (www.asthma.org.uk) recommends asthmatics to wash their beddings regularly, at 60 °C. This not only kills the mites, but it also removes their allergen, which is very soluble in water. Exposure to direct sunlight is also known to kill the mites, due to high temperatures and low RH. However, this technique can only be used in summer and it is more effective in certain climates. Electric blankets are also believed to kill some of the mites in a mattress, although they are unlikely to eradicate mites completely, since the heat does not penetrate the whole mattress. Steam cleaning, on the other hand, can be effective at both reducing mites and their allergens. Temperatures below zero for at least 24 hours (ideally 72 hours) can also be used to kill mites via liquid nitrogen or a conventional freezer. However, freezing does not remove mite allergens and therefore it should be coupled to vacuuming or washing.

Most control methods described so far can be more suitable for some objects (e.g. bedding) rather than others (e.g. mattresses). Furthermore, such methods require each object/surface to be treated individually and on a regular basis, which makes them time consuming and in some cases impractical. However, the microclimate of most mite habitats is strongly influenced by room conditions. Consequently, by controlling the hygrothermal conditions within buildings, it is possible to affect mite microclimates, therefore reducing/eradicating mite levels. Conceptually, this method uses similar principles as other hygrothermal control methods. In this thesis, the control of hygrothermal conditions in buildings for the purposes of eradicating mite infestations is referred to as "psychrometric control of house dust mites". This strategy does not immediately remove HDM allergens and it should therefore be coupled with an allergen-removing strategy, particularly if the dwelling is believed to be already infested by house dust mites. The psychrometric control is discussed further in next sections.

It should be mentioned that there is conflicting evidence as to whether mite control strategies can permanently reduce mite infestations to a level sufficient for health benefits. The Cochrane Review examined studies published until June 2004 in order to assess the effects of reducing exposure to house dust mite allergens in the homes of people with mite-sensitive asthma (Gøtzsche *et al.*, 2006). The reviewers selected 49 randomised controlled trials (2733 patients in total) of mite control measures with asthmatic people sensitised to house dust mites. The

reviewers concluded that "*Chemical and physical methods aimed at reducing exposure to house dust mite allergens cannot be recommended. It is doubtful whether further studies, similar to the ones in our meta-analysis, are worthwhile. If other types of studies are considered, they should be methodologically rigorous and use other methods than those used so far, with careful monitoring of mite exposure and relevant clinical outcomes*". It should be mentioned that the Cochrane meta-analysis only reviewed 2 papers using HDM psychrometric control (mechanical ventilation).

Many HDM control strategies are time consuming. As a consequence, they are mostly implemented by those mite sensitive individuals who are aware of their problem. However, the psychrometric approach could be "built-into" housing design or refurbishment. Therefore, as well as helping to alleviate existing symptoms, this could potentially prevent sensitisation to HDM allergen from occurring in the first place, and significantly save on the expense of treating allergic disease, which has been reported to cost the UK £2 billion a year (Chaytor, 2003). The next section illustrates the psychrometric control of house dust mites in more detail.

2.2.5 Psychrometric control of house dust mites

Food and space are usually abundant in mite microclimates, and predation and competition seem of little importance in the regulation of natural HDM populations. Hygrothermal conditions in mite microenvironments are therefore considered one of the primary determinants of HDM population growth (Hart, 1998). Hygrothermal conditions in mite microenvironments are affected by room conditions (Crowther and Wilkinson, *accepted for publication*). Consequently, many experts have advocated that controlling hygrothermal conditions in buildings – particularly reducing relative humidity – is a feasible way of reducing mite infestations (Arlian *et al.*, 2001a). Winter months are considered crucial for the reduction of mite populations in temperate climates, when the outdoor absolute humidity is rather low. If outdoor dry winter air is sufficiently heated in housing, the resultant indoor RH should be sufficiently low for mite growth to be inhibited. If enough mites die in winter, there won't be enough mites to

significantly replenish the population during the more favourable winter months (Crowther *et al.*, 2006).

A Danish study concluded that mite eradication occurs if indoor absolute humidity is kept below 7 g/kg (Korsgaard, 1998). However, Cunningham (1996) highlighted that this limit - often referred to as the "Korsgaard limit" - was originally intended for the Danish housing context, where indoor temperatures are rarely below 20-22 °C. Since mites adopt a hygroscopic solution to absorb moisture from the air (section 2.3.1) and since the Critical Equilibrium Humidity is temperature-dependant, *relative* humidity (as opposed to *absolute* humidity), as well as temperature, should be taken into account when trying to eradicate dust mites via psychrometric means. Taking into account the temperature dependency of CEH, Cunningham has suggested that relative humidity should be kept under 40% at 16 °C, 45% at 21 °C and 50% at 26 °C (Cunningham, 1996). Using Cunningham's figures, Lowe (2000) later demonstrated that in UK housing – which are often under-heated – HDM psychrometric control can only be achieved if internal temperatures are raised significantly. Lowe also points out that for warm dwellings (T=21 °C or more), HDM infestation could still occur, if ventilation rates are significantly less than 0.5 ach^{-1}.

Most studies examining the ideal hygrothermal conditions for HDM growth have been carried out with steady-state laboratory experiments. However, indoor hygrothermal conditions are variable, depending on outdoor conditions as well as on building characteristics and occupant behaviour. This makes it difficult, in some circumstances, to reduce relative humidity to levels which are consistently below CEH. For typical indoor temperatures, maintaining the average daily indoor RH below 50% is often recommended in order to reduce mite levels and their allergens (Arlian *et al.*, 2001a). However, some studies carried out experiments where mites were kept under varying hygrothermal conditions (Arlian et al., 1999a; de Boer *et al.*, 1998; Pike *et al.*, 2005). They found that mites were able to survive when exposed to brief spells of high RH, even though the daily average RH was below critical levels (de Boer *et al.*, 1998). However, some studies concluded that the reduction of indoor RH is still a viable control method, as mite development rates are much slower when HDM are only exposed briefly to favourable RH (Arlian *et al.*, 1999a). For example, Arlian found that maintaining

mean daily RH below 50% restricts mite population growth (*D farinae*) and allergen production, even if the RH rises above 50% for 2-8 hours daily. On the other hand, *D farinae* population growth can be completely prevented if the indoor RH is above 75-85% for 2 hours, provided that the indoor RH is kept below 35% for the remaining 22 hours (Arlian *et al.*, 1999b).

Although many experiments (mostly steady-state) have been carried out to assess how different combinations of temperature and RH affect mite populations, it is difficult to build a complete matrix of hygrothermal conditions and correspondent mite responses, since some hygrothermal conditions have yet to be investigated. Furthermore, the various experiments often adopted different methods, for example different diets. In addition, most experiments have been carried out by using mites which having been reared in laboratory conditions for many generations, may have become adapted to specific hygrothermal conditions and diet types. However, a study found that in temperate dry conditions eggs from a wild population were more resistant to mortality. This may have been because the laboratory population had become accustomed to the constant near-optimum conditions under which it was kept (Colloff, 1987). A more recent study on wild and laboratory-reared mites found that laboratory mites have stronger reproduction and development than wild mites, except when under environmental stress, and that diet (skin and dust vs. laboratory diet of dried liver and yeast) is a significant factor, particularly in sub-optimal conditions (Hart *et al.*, 2007). Therefore, caution should be taken when extending the findings from experiments on lab-mites to wild mite populations.

It should also be pointed out that higher feeding rates have been observed in mites held at higher RHs, which also result in higher faecal matter. A reduction of RH from 85% to 75% reduced feeding by 80% with a concurrent reduction in faecal matter in both DP and DF. RH also affects fecundity. Therefore, lowering the RH in homes – even if not below the CEH – may have a beneficial effect in reducing fecundity, mite population size and allergen load (Arlian, 1992).

In conclusion, feasible criteria for the psychrometric control of house dust mites in housing are still very much under discussion. A number of reasons contribute to this difficulty. Many experiments on hygrothermal conditions and house dust mites have been carried out under steady-state conditions using laboratory-reared

mites. Some studies have shown that wild mites may be more resilient to adverse hygrothermal conditions than laboratory-reared mites. Furthermore, some transient experiments suggest that mites might be able to survive under unfavourable hygrothermal conditions, provided that the indoor RH is favourable for a few hours every day. Nonetheless, in such conditions the growing rate of mite populations and their production of faecal matter are likely to be reduced. However, most experiments have only addressed changes in one hygrothermal variable (temperature or RH) at a time. In reality, changes in both temperature and humidity levels will occur, due to the interaction of weather conditions, building characteristics and occupant behaviour. Therefore, in real dwellings it can be difficult to reproduce those laboratory conditions which have been proven to reduce/eradicate mite infestations. On the other hand, it is also difficult to reproduce realistic transient conditions in a laboratory setting and assess their effect on mites. Furthermore, in a real setting issues such as a greater freedom of movement and food availability will affect mite distributions. In addition, some changes in hygrothermal conditions can result in unexpected dynamics in the mite population. For example, reductions in temperatures can result in increased relative humidity levels, which should be beneficial for mite growth. However, lower temperatures have also been proven to slow down the egg-to-adult development rate. This makes it difficult to assess the overall impact of such changes on mite populations. Consequently, a modelling approach has been advocated as a way to take account of the many variables, in order to formulate feasible strategies for the psychrometric control of house dust mites in specific contexts, such as the context of UK housing.

The next section discusses a number of intervention studies on HDM psychrometric control.

2.2.6 Intervention studies on HDM psychrometric control

The psychrometric control of house dust mites is based on the principle that reducing indoor moisture levels in dwellings should reduce mite infestation levels, due to the important role that relative humidity (and temperature) play in mite physiology. The feasibility of and the methods for HDM psychrometric control

partly depend on climatic conditions: in some climatic regions, psychrometric control may be more feasible than others. In temperate climates, winter months are considered crucial for the psychrometric control, since outdoor air has a lower absolute humidity (due to the lower external temperature), which can result in lower internal RHs if the outdoor air is sufficiently heated indoor. However, in winter low ventilation rates can occur, due to a reduction in window opening frequency, because of the colder outdoor air. Lower ventilation rates could in turn result in higher moisture levels, which would counteract the beneficial effect of lower outdoor absolute humidities. Indeed, some authors have postulated that the increase in asthma prevalence in the past decades is at least in part due to increased exposure to HDM allergens because of greater air-tightness levels imposed by concerns over energy efficiency (Howieson, 2005).

In temperate climates several studies have been carried out where mechanical ventilation (often coupled with heat recovery: MVHR) and/or dehumidifiers were utilised in order to reduce indoor humidity levels in winter and consequently mite concentrations. Some of these studies also tried to assess the (indirect) impact of the interventions on asthma symptoms. The use of mechanical ventilation has yielded successful results in some Scandinavian studies (Harving *et al.*, 1994a; Harving *et al.*, 1994b; Emenius *et al.*, 1998). However, the use of mechanical ventilation for the psychrometric control of house dust mites has resulted in some controversial results in the UK, whose more humid climate has been deemed by some authors as inadequate for a such strategy. A study conducted in the North-West of England on 18 houses – 9 with MVHR and 9 control houses – concluded that the MVHR unit does not reduce indoor humidity to levels capable of retarding mite population growth and decreasing mite allergens in the type of houses predominantly found in the mild and humid climate of the North-West of England (Fletcher *et al.*, 1996). In a successive study (Niven *et al.*, 1999), an additional central dehumidification modification of the MVHR (MVHRcd) was adopted, in order to further assess the viability of the psychrometric control of HDM in the UK. The researchers concluded that the MVHRcd system failed to confer a benefit in terms of mite allergen reduction, although it was not excluded that improvements to this technology might yield some positive results in future studies. However, as no measurements of air-infiltration were carried out, the

authors also pointed out that air-infiltration might have compromised the effectiveness of the MVHRcd system in some dwellings. In other UK studies mechanical ventilation appeared more successful for HDM control (McIntyre, 1992; Htut *et al.*, 1996; Warner *et al.*, 2000, Howieson *et al.*, 2003 and 2005). However, not all of the studies measured air-infiltration, nor the clinical efficacy of the remedial measures.

Howieson *et al.* (2003, 2005) examined the effect of a number of remedial measures (including MVHR, steam cleaning, new bedding) on 54 asthmatic subjects in North Lanarkshire. The study concluded that lung function measurements and health questionnaire data confirmed a significant improvement in the active group compared with the control group. However, the study presented a number of confounding variables. For example, no air-leakage tests were carried out. In addition, no skin prick tests were undertaken and consequently the project could not differentiate between the health effects influenced by a reduction in HDM allergen levels and/or the overall improvement on indoor air quality produced by greater ventilation rates. In another UK research project adopting MVHR, the homes of 40 mite-sensitive asthmatics in the Southampton area were randomised to receive a) mechanical ventilation with heat recovery (MVHR); b) high efficiency vacuum cleaners (HEVC); c) HEVC plus MVHR; d) no intervention (Warner et al., 2000). The patients were selected on the basis that their homes had characteristics suggesting low air leakage rates. In the homes with MVHR, tempered fresh air was supplied in the bedrooms. Homes and patients were monitored for 12 months, and changes in absolute humidity, mite numbers, Der p1 concentrations, lung function, bronchial hyperresponsiveness, and symptom scores were analysed. The results indicated that homes with MVHR achieved significantly lower absolute humidity levels than those without ($P<0.01$), with an associated reduction in mite numbers ($P<0.05$) and Der p1 concentrations ($P=0.006$) in bedroom floors. No significant reduction in mite numbers was found in the mattress, living room floor, or sofa. Some reductions were found in allergen concentrations in areas other than bedroom floors, although not statistically significant. However, since MVHR reduces live mites whilst allergen concentrations are reduced on a longer time scale, this was somewhat expected. There was a (non-statistically significant)

trend for MVHR units plus HEVC to be more effective than MVHR alone, which in turn was more effective than HEVC alone. Nevertheless, the reduction in allergen concentrations (most notable in bedroom floors) did not result in significant clinical improvements. However, the authors point out that the power of their study was low to detect clinical changes, and that this weakness was dictated by the cost of the MVHR units. Ventilating more areas of the dwellings was also suggested as a possible improvement to the study. The study was classified as "unclear" by the Cochrane review (Gøtzsche *et al.*, 2006). In other countries the use of MVHR has been considered inadequate for HDM control (e.g. New Zealand, Crane *et al.*, 1998).

Htut *et al.* (2001) carried out a UK double-blind trial on the homes of 30 asthmatics to determine the clinical efficacy of combined steam and heat treatment of home furnishings. For a subgroup of homes, the study also included a positive ventilation system (Nuaire) in the loft above the patient's bedrooms. The study concluded that a single heat-steam treatment of home furnishings reduced mite allergen levels, causing a significant improvement in bronchial hyperresponsiveness of asthmatic subjects. These improvements were sustained when a unit was installed to ventilate the patient's bedroom after the home had been steam-heat treated. However, this study was classified as "inadequate" in the Cochrane review (Gøtzsche *et al.*, 2006).

Mechanical ventilation is of course not the only method of implementing HDM psychrometric control. A UK study examined the effect of portable domestic dehumidifiers and of a behavioural programme on HDM counts and HDM allergen levels (Hyndman *et al.*, 2000). A total of 76 homes were randomly allocated to 3 groups, each receiving either: a portable domestic dehumidifier, a behavioural programme (e.g. airing the bed, keeping doors closed where moisture is being produced) or no intervention. The results indicated that neither the dehumidifier nor the behavioural intervention had a major effect on HDM counts o allergen levels. However, the authors pointed out that study did have a number of limitations. Firstly, the dehumidifier was not in operation for the winter period, and because of noise complaints it did not operate during the night. Secondly, a large number of households had no live mites, especially during the last measurements round. Thirdly, the sample size was relatively small, because of

financial constraints. A study carried out in the German Democratic Republic aimed to determine the effect of the installation of energy efficient measures on indoor climate, mite allergen (Der f1) and mould spore concentrations (Hirsch *et al.*, 2000). The bedrooms of 98 apartments were examined before and 7 months after installation of highly insulated windows and central heating systems. The results showed that the air exchange rate decreased from a geometric mean of 0.73 to 0.52 ach^{-1} (P=0.029). Temperature and absolute humidity increased but RH remained nearly unchanged. Der f1 concentrations increased on carpets (P<0.01) and beds (P=0.002). However, the study had a number of limitations. The authors themselves point out that the study lacked a control group and that discontinuous measurements of temperature and RH may have missed some significant hygrothermal data. Furthermore, the authors do no mention whether they measured outdoor hygrothermal conditions, nor whether they tried to assess how outdoor conditions affected changes in indoor conditions. Another method which could control indoor hygrothermal conditions is air conditioning. However, its effectiveness on HDM infestation is controversial (Crowther and Wilkinson, *accepted for publication*).

In summary, the efficacy of psychrometric methods for the control of HDM infestation in temperate climates is still under discussion. Although some studies have yielded some success in reducing mite and allergen levels, others were less successful and no study could prove a statistically significant improvement in asthma symptoms. However, since the principles of psychrometric control are based on sound evidence – i.e. the important role of hygrothermal conditions on HDM – the lack of evidence of its efficacy is most likely due to a number of issues:

a) Dust mites can survive spells of unfavourable hygrothermal conditions - particularly when these are alternated with brief favourable spells. At present, it is difficult to control such brief spells of elevated RH in dwellings and it is unclear to what extent and how tightly such spells need to be controlled, in order to reduce mite infestation levels. A modelling approach – taking into account all the variables influencing HDM growth - is probably the only way of moving forward in this impasse.

b) Mechanical means (e.g. mechanical ventilation) are often considered the ideal psychrometric control methods, since they potentially guarantee a tighter and more restricted control of indoor hygrothermal conditions. However, these methods are expensive; furthermore, they cannot be applied in all circumstances. For example, mechanical ventilation with heat recovery is more effective in airtight dwellings and involves a large amount of ductwork. The impact of such devices is dependent on the dwelling characteristics and the occupant behaviour. In order to control for this confounding factors, a greater sample size is required. Consequently, most studies aiming to assess the efficacy of mechanical means often lack the sample size required to detect statistically significant improvements in HDM levels and/or asthma symptoms.

c) Any study aiming to assess the efficacy of psychrometric control methods on HDM infestation levels and on asthma symptoms is faced with a very large number of interrelated factors, all of which should ideally be measured at some level. The length of the study can also be crucial. However, this translates in very high costs and most studies had to choose between the number of measured variables and the number of cases under scrutiny. However, so far no "magic formula" for study design has been found. Faster, cheaper and more reliable methods are needed for: assessing levels of mite infestation and of mite allergen exposure, measuring changes in health outcomes, measuring ventilation rates, assessing levels of exposure to other indoor pollutants such as moulds, endotoxins, etc.

d) Many intervention studies on the efficacy of HDM psychrometric control have been carried out by epidemiologists with insufficient knowledge of building physics or by building scientists with insufficient knowledge of HDM biology or asthma. This often resulted in flawed study design and/or data analysis. However, although a multidisciplinary approach is desirable, this is often hindered by difficulties in obtaining funds since it becomes unclear which research council or governmental body should fund the research.

Due to the above difficulties in devising and funding intervention studies on HDM psychrometric control, it is perhaps unsurprising that not many of such studies have been carried out in recent years.

2.3 Modelling hygrothermal conditions in beds and their effect on HDM populations

The previous sections highlighted that temperature and relative humidity play an important role in house dust mite physiology, and that mattresses are a crucial source of HDM allergens. By controlling the hygrothermal conditions experienced by house dust mites in beds, the reduction/eradication of mite infestations can be potentially achieved. Hygrothermal conditions in beds are affected by room conditions, mattress and bedding properties, and by the bed's occupant (e.g. length of bed occupation, heat and moisture output). In order to be able to control the bed microclimate, it is necessary to fully understand the interactions between all these variables. Furthermore, various combinations of temperature and RH affect mite populations differently. Therefore, a modelling approach is useful for 1) predicting the effect of bedroom conditions on beds (bed model); 2) predicting the effect of bed conditions on mite populations (population model).

This section reviews the issues associated with modelling hygrothermal conditions in beds, as well as modelling the effect of hygrothermal conditions on HDM populations. In particular, the following issues are addressed:

- In order to model hygrothermal conditions in beds, heat and moisture transfer rates must be calculated, in relation to given boundary conditions. The principles of heat and water vapour diffusion in porous materials are discussed in section 2.3.1.
- The rate of heat and moisture transfer in a bed is also affected by its material properties, which are discussed in section 2.3.2 – with a focus on textiles.
- The boundary conditions on a bed surface are determined by the room conditions and by the bed occupant. When the bed is occupied, the boundary conditions of the bed's top surface are affected by the heat and moisture produced by the bed occupant. This is discussed in section 2.3.3. The room conditions, on the other hand, can be measured or predicted. Several models have been developed over the years for the prediction of indoor conditions in buildings. A detailed review of the available building models exceeds the scope

of this thesis. However, it should be mentioned that to accurately predict the relative humidity within the bedroom, the simulation package must adequately account for ventilation, moisture production and ideally also for the buffering effect of hygroscopic materials in walls and furniture, on the relative humidity of the room (Ridley *et al.*, submitted). Currently, the IEA *Annex 41* (http://www.ecbcs.org) is working to acquire a better knowledge of the whole building heat, air and moisture balance.

- Section 2.3.4 discusses existing hygrothermal models of beds (other than the models tested in this thesis, which are described in Chapter 3).
- Section 2.3.5 discusses existing models of the impact of hygrothermal conditions on mite populations (other than the models tested in this thesis, which are described in Chapter 3).
- Section 2.3.6 discusses methods for model validation.

2.3.1 Heat and water vapour diffusion in porous materials

For given boundary conditions, hygrothermal conditions in a mattress are determined by the amount of heat and moisture transfer through the mattress itself. This section is a brief overview of heat and water vapour transfer via conduction/diffusion – since the hygrothermal models discussed in this thesis do not explicitly model other heat and moisture transfer processes such as radiation, convection, liquid water diffusion or capillary suction. Numerical modelling is also briefly discussed, with a focus on the explicit finite-difference approach, which is utilised in the model Lectus.

It is possible to establish the temperature distribution within a medium resulting from conditions imposed on its boundaries, by referring to the heat diffusion differential equation. It can be demonstrated that the heat diffusion differential equation[4] is obtained by referring to the conservation law and to Fourier's law (Incropera and DeWitt, 1996). In relation to an infinitesimally small control volume (dxdydz), the **heat diffusion differential equation** is:

[4] This equation is referred by other authors as the general (or governing) differential equation for heat transfer

$$\frac{\partial}{\partial x}\left(k_x \frac{\partial T}{\partial x}\right)+\frac{\partial}{\partial y}\left(k_y \frac{\partial T}{\partial y}\right)+\frac{\partial}{\partial z}\left(k_z \frac{\partial T}{\partial z}\right)+\dot{q}=\rho c_p \frac{\partial T}{\partial t} \qquad [2.3.1]$$

where $\dot{q}$ is the heat generated within the control volume. The term $\rho c_p \frac{\partial T}{\partial t}$ in [2.3.1] refers to the heat stored in the medium, where ρ is the material's density (kg/m^3) and c_p is its specific heat capacity (J/kgK). The remaining terms of equation [2.3.1] refer to the *net* conduction heat flux, in the direction of the flux. For example, k_x is the thermal conductivity (W/mK) in the direction x.

If the thermal conductivity is a constant k and with no heat generation, equation 2.3.1 becomes:

$$\frac{\partial^2 T}{\partial x^2}+\frac{\partial^2 T}{\partial y^2}+\frac{\partial^2 T}{\partial z^2}=\frac{1}{\alpha}\frac{\partial T}{\partial t} \qquad [2.3.2]$$

where $\alpha=k/\rho c_p$ is the *thermal diffusivity* (m^2/s).

It can be demonstrated that the **water vapour diffusion differential equation** is analogous to the heat diffusion differential equation (equation 2.3.1). Assuming no generation of water vapour within the volume and with constant density and diffusion coefficient, the equivalent of equation 2.3.2 for water vapour transfer is:

$$\frac{\partial^2 P}{\partial x^2}+\frac{\partial^2 P}{\partial y^2}+\frac{\partial^2 P}{\partial z^2}=\frac{1}{D}\frac{\partial P}{\partial t} \qquad [2.3.3]$$

where D is the **moisture diffusivity** (m^2/s), which in the hygroscopic range is the ratio between vapour permeability and volumetric moisture capacity. Outside that range, the moisture diffusivity is the ratio between moisture permeability and volumetric moisture capacity.

Methods for solving the heat and the water vapour diffusion differential equations include the use of analytical, graphical and numerical (finite-difference, finite-element and finite-volume) approaches. For 2-dimensional and 3-dimensional heat and moisture transfer problems, solving the partial differential equation analytically is complicated and may be obtained for only a restricted set of simple geometries and boundary conditions not normally found in dwellings. In contrast to analytical methods, which provide *exact* results at *any* point, the graphical and numerical methods can provide only *approximate* results at *discrete* points.

However, because the methods can often accommodate complex geometries and boundary conditions, they are often preferred. Since a numerical solution enables determination of the temperature at only *discrete* points, it is necessary to identify such points by subdividing the medium of interest into a number of small regions, each of which are represented by a reference point (nodal point) at its centre. The temperature in each node is a measure of the average temperature of the region. In addition to being discretized in space, the solution also requires that the problem is discretized in time. The accuracy of the finite-difference solution may be improved either by decreasing the size of the cells (Δx) or the time step (Δt). However, since the number of nodes increases with a smaller Δx, and the number of time intervals required to carry out the solution to a prescribed final time increases with a smaller Δt, computation time increases. The choice of Δx is typically based on a compromise between accuracy and computational requirements. Once this selection is made, the value of Δt may not be chosen independently. An undesirable feature of the explicit method is that it is not unconditionally stable, since numerically-induced oscillations may occur, which are physically impossible. To prevent this problem, the time step Δt must be maintained below a certain limit and this dependence is called *stability criterion*. For heat transfer, it can be demonstrated that the stable time step is directly proportional to the heat capacity and to the dimension of the cells, and inversely proportional to the thermal conductivity. A similar relationship occurs for water vapour transfer (Incropera and DeWitt, 1996). The *implicit* finite-difference method is unconditionally unstable (no restrictions on Δx and Δt; potentially less computational time). However, in the implicit method reasonable values must still be assigned to Δx and Δt in order to obtain accurate results, and the method can be more difficult to solve (Incropera and DeWitt, 1996).

2.3.2 Hygrothermal properties of porous materials

As illustrated in the previous section, the rate of heat and moisture transfer in a porous medium is dependent on the hygrothermal properties of the medium. In particular, in order to solve the heat and the water vapour diffusion differential equations (see previous section), it is necessary to know a material's: density, thermal conductivity, specific heat capacity, water vapour permeability, and

specific moisture capacity. In this section, these properties are defined and discussed, including methods for testing such properties. Since the mattress is mainly constituted of textile materials, a particular focus will be given to textiles.

A material's **density** is the ratio between its mass and its volume (kg/m^3), and it is therefore relatively easy to measure. Usually, a dry sample it utilised (*dry* density). The **thermal conductivity** k (W/mK) of a material can be defined as the quantity of heat, *Q (J)*, transmitted in time *t* (s) through a thickness *L (m)*, in a direction normal to a surface of area *A* (m^2), due to a temperature difference ΔT *(K)*, under steady state conditions and when the heat transfer is dependent only on the temperature gradient. Therefore:

thermal conductivity = heat flow rate × distance / (area × temperature difference)

$$k = \frac{Q}{t} \times \frac{L}{A \times \Delta T} \qquad [2.3.4]$$

The transmission of heat through a fabric occurs both by conduction through the fibre and the entrapped air and by radiation. Practical methods for testing the thermal conductivity (e.g togmeter and guarded hotplate: Saville, 1999) measure heat transmitted by both mechanisms. It is important to measure thermal conductivity at temperatures which are likely to be encountered in use, since thermal conductivity varies with temperature. Furthermore, thermal conductivity increases with an increase in the material's moisture content.

The **specific heat capacity** (J/kgK) of a material is defined as the thermal energy required to increase the temperature of a unit mass of a material by 1 K. A material's specific heat capacity can be measured using a calorimeter.

The **vapour permeability** μ_x ($kg \cdot m^{-1} \cdot Pa^{-1} \cdot s^{-1}$) of a material can be defined as the quantity of water vapour *m (kg)*, transmitted in time *t* (s) through a thickness *L (m)*, in a direction normal to a surface of area *A* (m^2), due to a vapour pressure difference ΔP *(Pa)*, under steady state conditions and when the heat transfer is dependent only on the pressure gradient.

Vap. permeability = vapour flow rate × distance / (area × pressure difference)

$$\mu = \frac{m}{t} \times \frac{L}{A \times \Delta P} \qquad [2.3.5]$$

If the gradient in equation 2.3.5 is expressed in terms of vapour *concentrations* (kg/m^3) - rather than vapour *pressures* (Pa) - the vapour permeability has the unit of m^2/s. The permeability to vapour of a specimen is sometimes also expressed in terms of vapour resistance factor or as vapour diffusion thickness. The **vapour resistance factor** (dimensionless, sometimes called s_d-*value*) is defined as the ratio between the vapour permeability of stagnant air and that of the material under identical conditions (Kumaran, 1996). The **vapour diffusion thickness** (m) is the product of a specimen's thickness and its vapour resistance factor (Kumaran, 1996).

Two main methods are available for measuring the water vapour permeability of a test fabric: the cup method and the sweating guarded hotplate method. The first method can be either a wet cup or a dry cup. The results from the wet and the dry cup methods can be quite different, particularly for hygroscopic materials such as wood. This is because the 2 methods test the fabric under 2 different humidity ranges (Eastop and Watson, 1992). The air-gaps above and below the test fabric in the cup methods affect the test results, due to the vapour resistance of the air. This is particularly problematic for highly permeable materials. Therefore, vapour permeability can also be tested with the **inverted cup method**, so that the water is in contact with the inner surface of the fabric, and the air gap is removed. This type of test tends to give more favourable results for hydrophilic films (Saville, 1999). A variation of the inverted cup method is the **desiccant inverted cup method**, where the fabric is between water and a desiccant solution. The upright cup methods simulate the situation in which the skin is at normal or above normal hydration levels and the fabric layers are dry, while the inverted cup methods appear to more closely simulate the situation in which a fabric is in direct contact with a saturated layer (McCullough *et al.*, 2003).

Vapour permeability in a material changes with changes in hygrothermal conditions. Vinha *et al.* (2002) found that the water vapour resistance of a material is a function of ambient RH, where in general vapour resistance is lower at higher RHs. However, the impact of RH on the resistance varied in different materials. Differences in vapour permeabilities can be found even when using the same test method. For example, the vapour permeabilities of 3 different building materials (hard wood fibre board, underlay for roofing, polyethylene damp-proof

course) were measured with a standardised wet cup method by 5 different laboratories (Nordtest, 2003). It was found that the water vapour resistance and the s_d-value can be identified with a certainty of +/- 10%. This is if the resistance of the air layer is corrected for, the air velocity above the test sample is between 1.5 and 2.0 m/s and the masked edge effect is corrected for.

McCullough *et al* (2003) compared the vapour permeability of 26 different waterproof, windproof and breathable fabrics measured using different standard methods (wet cup, inverted cup, desiccant inverted cup, sweating guarded hotplate). They found that the permeability was consistently highest when measured with the desiccant inverted cup, followed by the inverted cup and upright cup. With the exception of the inverted cup method, most methods were statistically correlated. The desiccant inverted cup method and the sweating guarded hotplate were highly correlated. McCullough *et al.* concluded that permeability varies with test method, material's type and test conditions. Therefore, samples should be tested with the method and conditions that are closer to their end use.

The **specific moisture capacity** (kg/kgPa) is defined as the increase in the mass of moisture in unit mass of material that follows an increase in vapour pressure or suction (Kumaran, 1996). In order to assess a material's specific moisture capacity, it is necessary to measure its **moisture content** under different environmental conditions. Moisture content (dry) is expressed in terms of mass of moisture per unit volume of dry material (kg/m^3), or mass of moisture per unit mass of dry material (kg/kg). For a given material, the moisture content is governed by: ambient RH, ambient temperature, amount of time exposed to specific ambient conditions, and the previous history of the sample (because of the hysteresis: Trechsel ed, 1994). Different methods are available to determine a material's sorption isotherms. Most methods involve exposing the dry material to different RHs – usually with salt solutions or climatic chambers – and weighing the sample accordingly. For example, the automated sorption balance is a microbalance in a carefully-controlled climatic chamber. The limitation of this method is that it requires a small size for the test sample (Svennberg, 2003).

Since sprung mattresses include an air layer, it should be mentioned that in addition to heat conduction, heat transport also occurs via convection and

radiation in an air layer. Similarly, in addition to water vapour diffusion, water vapour can also be transferred by convection in an air cavity. Although many heat and moisture transport models do not simulate convection and radiation *explicitly*, the additional transport mechanisms (convection, radiation) in an air cavity are indirectly included when measuring the thermal resistance and the diffusion resistance of the air cavity (CIBSE, 1986). The relative contributions of heat conduction, convection and radiation in an air cavity depends on: the surfaces' emissivity, the dimensions of the air cavity, the direction of the heat flow (horizontal/vertical), and the temperature difference between the two surfaces (the latter is usually ignored in building physics). Unless the surfaces of the air cavity are metallic, their emissivity is assumed as high (CIBSE, 1986). Experiments are usually carried out in order to determine the thermal and vapour diffusion resistances of air cavities (usually unventilated) with specific characteristics (e.g. horizontal/vertical, specific thickness, etc.). The thermal conductivity and vapour permeabilities obtained via these experiments include the effect of convection and radiation in air cavities with similar characteristics to those tested.

Finally, it is worth mentioning that the hygrothermal properties of mattresses, beddings and their components are not all easily available in published data. Svennberg *et al.* (2006) studied 2 bed systems, one solely made of homogenous polyether foam, the other mattress with springs and an overlay of polyether foam, covered on both sides with wool batting. The properties of these 2 mattresses were determined as a combination of measured and published data, as illustrated in Table 2.3.1. Svennberg *et al.* also point out that if the compression and the 3-dimensional deformation of the mattress during use were to be considered, this would add an additional challenge in the determination of the properties. The properties of the mattress used in the Series 2 fieldwork of this thesis were measured by the *Performance Clothing Research Group*, at the *Centre for Technical Textiles*, University of Leeds. They are provided in Chapter 5.

Table 2.3.1 Properties of some mattress and bedding materials (Svennberg *et al.*, 2006)

	Heat Conductivity* (W/mK)	Specific Heat Capacity (J/kgK)	Vapour Permeability (m^2/s)	Moisture Capacity (kg/m^3)
Polyether foam	0.040	1007	(25.9×10^{-6})#	1.2#
Wool batting	0.039	1008	19.5×10^{-6}	9.5#
Polyester batting	0.039	-	-	-
Cotton sheet	0.044	-	(81×10^{-6})#	134#

*In the original paper, the "Heat Conductivity" is provided, in W/m^2K. In this thesis, it is assumed that "heat conductivity" stands for thermal conductivity (W/mK) and that there was misprint in the units provided in the original paper. #Measured by Svennberg *et al.*

In summary, the rate of heat and moisture transfer in a porous medium is dependent on the hygrothermal properties of the medium. These properties can be measured with different test methods. However, hygrothermal properties can change according to different test methods and ambient hygrothermal conditions. Consequently, samples should be tested with the method and conditions that are closer to their end use. If an object is likely to experience a wide range of hygrothermal conditions in its use, ideally a sample should be tested under most of these conditions. Compression and geometric deformations usually encountered in mattresses during use add a further problem in the determination of the mattress properties.

2.3.3 Hygrothermal conditions in mattresses: thermoregulation and sleep

Heat and moisture outputs from the human body affect the hygrothermal conditions in a bed, particularly when the bed is occupied. They have an impact on the boundary conditions which should be considered when modelling a bed. In this section, the thermoregulatory mechanisms occurring in the human body are discussed, with a focus on thermoregulation during sleep.

In humans, heat must be exchanged so that the internal body core temperature is maintained at a level of approximately 37 °C. Heat is exchanged with the surroundings via conduction, radiation, convection and evaporation.

It can be assumed that for long exposures to a constant (moderate) thermal environment with a constant metabolic rate, a heat balance will exist for the human body, following the equation (Fanger, 1970):

$$H-E_d-E_{sw}-E_{re}-L=R+C \qquad [2.3.6]$$

where H is the body's internal heat production; E_d is the heat loss by water diffusion through the skin[5]; E_{sw} is the heat loss by evaporation of sweat from the surface of the skin; E_{re} is the latent respiration heat loss; L is the dry respiration heat loss; R is the transfer from the skin to the outer surface of the clothed body (conduction through clothing); C is the heat loss by convection from the outer surface of the clothed body. All the terms in equation 2.3.6 are in kcal/hr.

The heat balance in the human body is therefore affected by: the internal heat production of the body, the thermal resistance of the clothing, the temperature and the pressure of water vapour in the ambient air, the mean ambient radiant temperature, the ambient air velocity, the mean skin temperature, and the heat loss by evaporation of sweat secretion. For a given level of activity, the skin temperature and the sweat secretion are the only physiological variables influencing thermal comfort (Fanger, 1970). The mean skin temperature decreases with increases in activity levels, and it varies from 34-35 °C for sedentary activity (50 kcal/hr m^2) to 31 °C for more intense activities (150 kcal/hr m^2). At high activity levels, sweating takes place (Fanger, 1970).

Heat balance in the human body is easily maintained in the thermoneutral zone, defined as the range of air temperatures within which the metabolic rate is minimum and thermoregulation can be achieved by non-evaporative processes. Outside the thermoneutral zone, the body temperature slightly rises (warm environment) or falls (cold environments), and at the same time sweating or metabolic heat production increases, to prevent further changes in body temperature (Bach *et al.*, 2002). Whilst the body core is homeostatically regulated at around 37 °C, the body "shell" is not and its temperature depends largely on the environmental temperature. The body shell is constituted of 2 components: the distal skin regions (e.g. hands and feet), and the proximal skin regions (e.g. torso). A specialised thermoregulatory system is localised in the distal skin regions: the arteriovenous anastomoses (AVAs), which act as shunt for rapid blood flow

[5] Water vapour diffusion through the skin is one part of the insensible perspiration (the other is respiration, a process which occurs independently from thermoregulatory control). The magnitude of the diffusion per unit area of the body is assumed to be proportional to the difference between saturated water vapour pressure at the skin temperature and the partial pressure of water vapour in the ambient air (Fanger, 1970).

enabling rapid heat exchange. When the AVAs are open, heat exchange is about 10,000 times faster than capillary blood flow. During exposure to cold, peripheral blood flow decreases, in order to reduce heat loss from the body core. Shivering may also occur in adults, which increases metabolic heat production. On the other hand, during exposure to heat, blood vessels are dilated, in order to increase heat loss, which if necessary may be enhanced by sweating (Kräuchi and Wirz-Justice, 2001).

The set-point for the thermoregulation of the human body is not constant, but fluctuates according to endogenous factors, such as: age, vigilance levels, sleep deprivation, thermal adaptation, fever, intake of food or fluids, posture during sleep, heat exposure before sleep, depression, etc. (Bach *et al.*, 2002). Furthermore, the core body temperature (CBT) is not constant throughout the day, following a circadian rhythm. When the sleep-wake cycle is synchronised with the light-dark cycle, the maximum CBT occurs in the early evening, and the minimum in the second half of the nocturnal sleep episodes (Kräuchi and Wirz-Justice, 2001). Sleep is usually initiated on the declining portion of the CBT curve when its rate of change – and body heat loss – is maximal. In the morning, when heat production is dominant over heat loss, CBT increases and so does the propensity to wake-up. It has been shown that distal skin temperature rises in the evening, whereas heat production, proximal skin temperature and CBT decline, and in the morning the inverse occurs. This inverse circadian regulation of distal and proximal skin temperature is an index of a circadian regulation of the 'core/shell' ratio: i.e. in the evening the shell is "larger" due to vasodilation. Kräuchi hypothesises that both the evening increase in sleepiness and the exponential decline of sleepiness upon awakening can be described as a function of changes in distal vasodilation. (Kräuchi, 2006).

Figure 2.3.1 shows variations in CBT, proximal and distal skin temperatures before and during sleep, or without sleep.

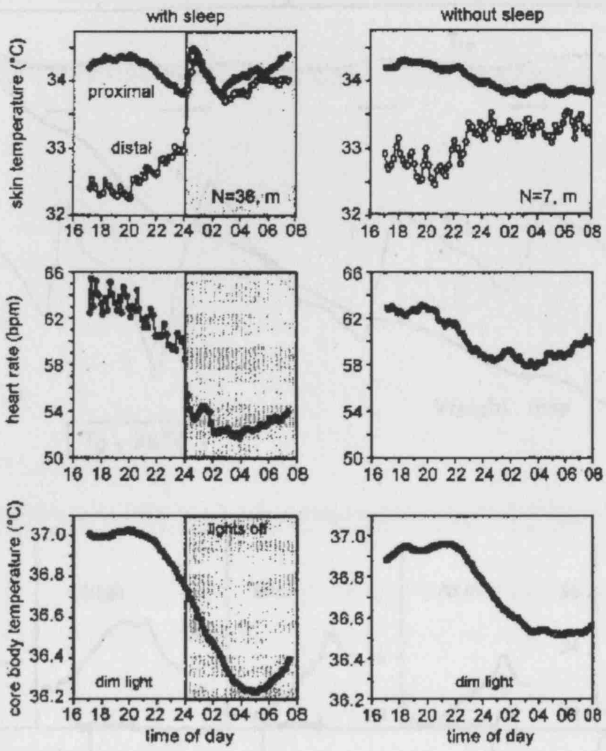

Fig 2.3.1 Changes in core body temperature, skin temperature, and heart rate, with or without sleep (Kräuchi and Wirz-Justice, 2001).

Henane *et al.* (1977) measured the variations in evaporation and body temperatures during sleep in 3 healthy male subjects (age: 24, 25, 27). The results showed variations in temperature and evaporation, characterised by 2 rhythms: a basal circadian rhythm (long-lasting) and superimposed on it, a short-lasting rhythm (80-90 minutes), conditioned by the occurrence of REM phases[6] of 10-20 min. These short-term waves reached an amplitude of 0.5-2 °C in the skin temperatures. Figure 2.3.2 shows the changes in body temperatures in a subject under warm conditions (35 °C).

[6] Human sleep is characterised as REM sleep (Rapid Eye Movements) and non-REM sleep. The Non-REM sleep is divided into 4 stages, of which stage 3 and 4 are called deep sleep, or slow-wave sleep (SWS). In humans, REM and Non-REM sleep alternate (Okoda *et al.*, 2005).

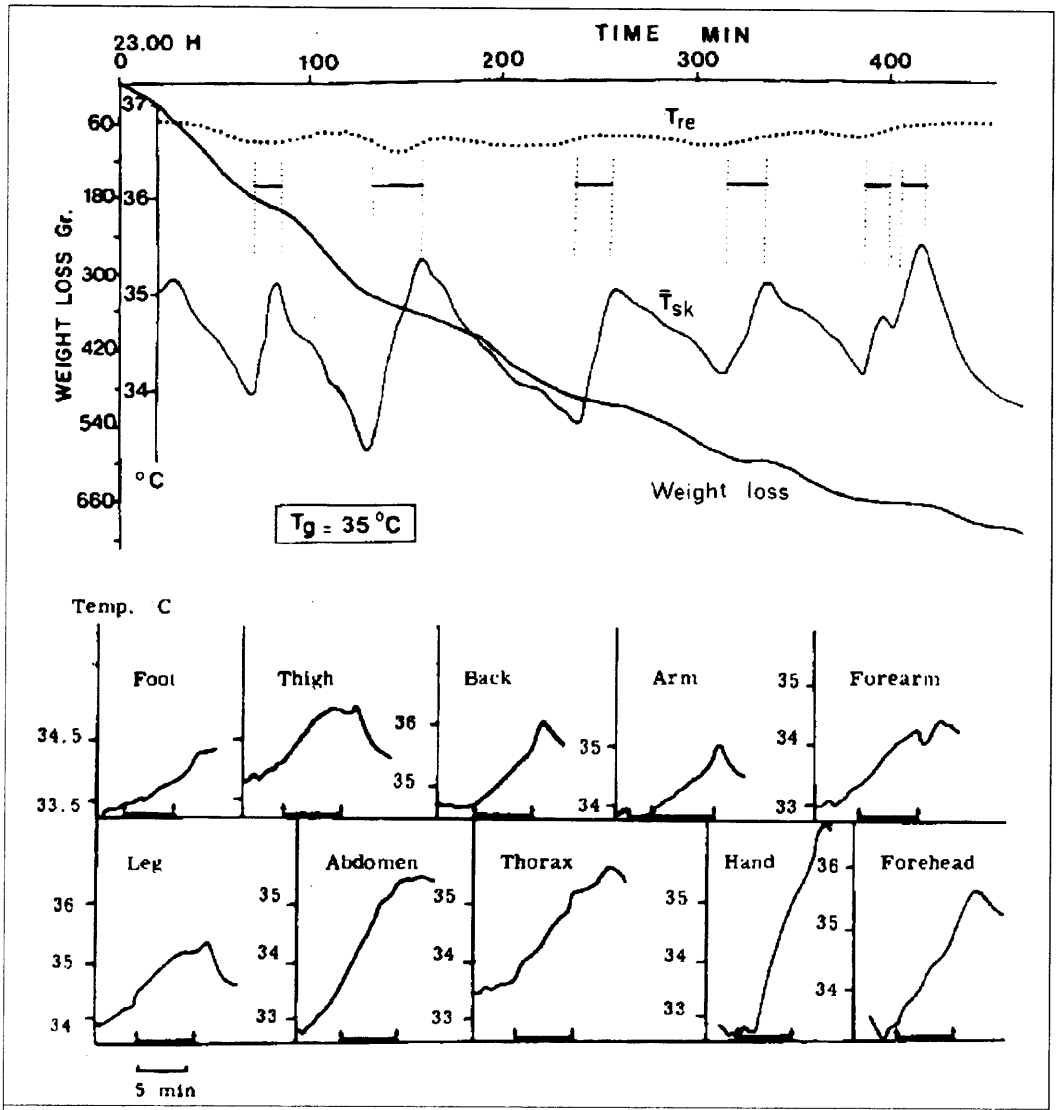

Fig 2.3.2 Changes in core body temperature and skin temperature during sleep, with an ambient temperature of 35 °C (Henane *et al.*, 1977).

Figure 2.3.3 shows the mean evaporative rate for a range of ambient temperatures.

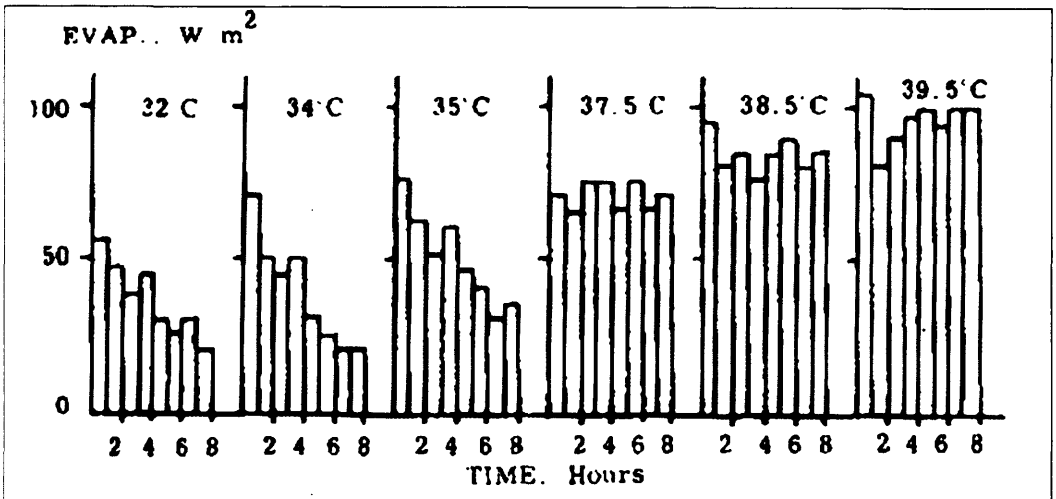

Fig 2.3.3 Mean evaporative rate during sleep under different ambient temperatures (Henane *et al.*, 1977).

Henane *et al.* (1977) also observed that water intake during the night induces a sudden increase in skin evaporation with a reduction of skin temperature.

As already highlighted above, sleep, thermoregulation and ambient conditions are interrelated, and thermoregulation mechanisms can change according to various sleep phases and to ambient conditions (Henane *et al.*, 1977). On the other hand, several studies have shown an effect of ambient conditions on sleep quality – although many of such studies have adopted rather high temperatures (>30 °C). There is some evidence that cold environments are more disruptive to sleep than warm ones. Furthermore, decreasing rather than increasing changes in temperature cause more disturbance, but these disturbances are not observed when exposed to lower rate of temperature changes (0.027 °C/h instead of 1 °C/h). There is also some evidence that ambient temperature during wakefulness may affect later sleep (Bach *et al.*, 2002).

This section illustrated the thermoregulatory mechanisms occurring in the human body, with a focus on thermoregulation during sleep. The human body has a number of mechanisms to maintain its core temperature at approximately 37 °C. These mechanisms include vasodilation, sweating, shivering, changes in metabolic rate, etc. The skin temperature is approximately at 34 °C. However, the set-point for the thermoregulation of the human body is not constant, but fluctuates according to endogenous factors, such as: age, vigilance levels, sleep deprivation, thermal adaptation, fever, intake of food or fluids, posture during

sleep, heat exposure before sleep, depression, etc. Furthermore, body core and skin temperatures vary according to circadian rhythms and to the sleep phase. Variations of 0.5-2 °C have been observed during sleep in skin temperatures, particularly for the distal skin areas. The rate of evaporation is also affected by sleep phases, as well as by room conditions. Since most studies on sleep and hygrothermal conditions have been carried out under laboratory-controlled conditions, at present it is difficult to estimate the magnitude of variations in heat and moisture outputs *across* individuals, and *within* individuals under realistic ambient conditions.

2.3.4 Existing models of hygrothermal conditions in beds

Hygrothermal conditions in beds can be very variable, depending upon a number of interacting factors, such as: climate; building characteristics (especially insulation and air-tightness); heating and ventilation patterns; moisture production; type of mattress; length of time the mattress is occupied, etc. By establishing how such factors interact and affect mite survival, it might be possible to identify those building features and occupant behaviours influencing mite growth. In this section, existing models for the prediction of hygrothermal conditions in beds are discussed, as well as models predicting the impact of hygrothermal conditions on mite populations. The models tested in this thesis are described in the following chapter.

In the mid-90s, Cunningham was amongst the first building scientists to emphasize the potential hygrothermal differences between room conditions and mite microclimates, and to call for a deeper understanding of these differences (Cunningham, 1996 and 1998). Cunningham also highlighted the need to develop a physical microclimate model coupled with a dust mite population model. In fact, he developed a mite population model, which is discussed later in this section. Cunningham also developed a heat and moisture transfer model for mite microclimates (Cunningham, 2004). Cunningham's hygrothermal model is a numerical, transient one-dimensional model, which can include room air as well as other room components and mechanical ventilation. Central to the model is a one-dimensional 'slab' in which the microenvironment of interest is modeled. The

slab may model, for example bedding or a mattress, a carpeted floor, a wall, or a piece of furniture. It may be multilayered with properties that may be either constant or variable. Heat flow through the building envelope can be modeled, either by neglecting thermal storage, or by use of the response factor method. Active components can include furnaces, humidifiers and dehumidifiers, and other air conditioning equipment. Both vapour and liquid moisture movement are taken into consideration. Cunningham compared his bed model's predictions with data resulting from the monitoring of a real bed over 24 hours. The bed was modeled with the bed occupant being simulated by having one layer producing heat at the rate of 36 W and moisture at 0.02 l/h. Cunningham concluded that good agreement existed between monitored and predicted results. He also added that a closer agreement would require more accurate properties of the bedding materials, and modeling the convective and radiative transfer in the internal cavity of the inner sprung.

Svennberg *et al.* (2006) examined two real mattresses (foam and sprung), whose hygrothermal conditions were measured for 4 days (sleeping period: 8-10 hours). The beds were occupied at night by participants in pre-adolescent age. Considerable variations were found in the RH monitored during the day (unoccupied beds) between the 2 mattress types. Svennberg *et al.* then compared the measurement results with the predictions of a transient one-dimensional heat and vapour model. Constant materials properties were used (Table 2.4.1) and the temperature dependency for the moisture parameters neglected. Measured room conditions were used for the boundary conditions, but when the bed was occupied the top surface of the mattress was set at a temperature of 35.5 °C (which reproduced measured values), with a surplus vapour content of 14 g/m^3. The surplus vapour content was derived from the estimation of 20 g/h of insensible moisture production, half of which would affect the mattress. Svennberg *et al.* concluded that there was a reasonable agreement between the model and the measured results. However, the authors also highlighted the need for more refined material properties, which for example take into account both the temperature and moisture dependency, and possibly the effect of compression. The authors also suggest further research on the importance of sleeping patterns and of individual moisture production rates.

2.3.5 Existing models of the impact of hygrothermal conditions on mite populations

Temperature and RH play an important role on mite physiology. However, currently there are not many models available, which predict the effect of hygrothermal conditions on mite populations. The only published mite population model - apart from the two population models tested in this thesis – was developed by Cunningham (Cunningham, 2000). Cunningham collated the available published information on HDM population's doubling/halving times under different steady-state hygrothermal conditions (Cunningham, 1996). Since information was not complete for any single HDM species, Cunningham put together information from experiments using 3 different species (DF, DP, EM) and assumed that population grows exponentially on a daily basis. In order to establish the dividing line between HDM population growth and decline, Cunningham also assumed that population growth (or decline) occurs above (or below) the Critical Equilibrium Humidity, which he obtained by curve-fitting published values for the steady-state Critical Equilibrium Humidity (CEH) of DF mites in relation to different temperatures (T), obtaining the equation:

$$CEH=56.75-0.9917T+0.05T^2-0.0003T^3 \quad [2.3.7]$$

By curve-fitting the published information on population halving/doubling times, Cunningham obtained the following equations:

$$\text{If } RH>CEH, \text{ then } Growth=1+4.9\times10^{-5}T(RH-CEH) \quad [2.3.8]$$

$$\text{If } RH<CEH, \text{ then } Decline=1-3.38\times10^{-4}T(RH-CEH) \quad [2.3.9]$$

where "Growth" and "Decline" are population multiplication factors per day (e.g. 1.1 represents 10% population growth per day).

Cunningham's was the first model of its kind which could be used to predict the effect of different combinations of temperature and RH on HDM growth. However, Cunningham's model is not validated as such, and it does not apply to a specific mite species, since data was utilised which derived from experiments on different HDM species. Furthermore, in Cunningham's model there are no constraints on mite growth at high RHs - which are known to occur in reality. This

is because the data that Cunningham used did not include extreme hygrothermal conditions. The main limitations of Cunningham's population model arise from the lack of available information on the effect of a large range of hygrothermal conditions on HDM populations. Furthermore, since he had to collate data, methodological differences may have influenced the results. The MPI model – described in the next chapter – tries to overcome part of these problems, by using data from experiments which were all carried out by the MPI's authors, in a consistent fashion and for a single species (DP).

2.3.6 Model testing and validation

In order to utilise any model adequately, it is necessary to assess its limitations, its uncertainties and its ability to represent the modelled phenomena. In this section, the issue of model testing and validation is discussed.

A number of different methods are available to test and validate models. These methods can be grouped into 5 categories (Crawley, 2001):

1. Analytical tests, which compare against mathematical solutions;
2. Comparative test, which compare against other software;
3. Empirical tests which compare against experimental/field data;
4. Sensitivity tests, which compare small input changes versus a baseline run;
5. Range tests, which exercise the program over a wide rage of input variables (usually to identify any bugs in the program).

A number of analytical tests currently exist for building simulation programs, where instructions are provided for the simulation of a standard building with specific characteristics. The reference results are also provided, which have to be compared with the results from the model being tested. These results can also be tested against those provided by other models (Crawley, 2001). However, since bed and population models are few in numbers and relatively new, no standard analytical or comparative test is currently available for these models. Consequently, at present the validation of bed and population models has to be empirical. This consists in the comparison between predicted and measured results. In most cases, simulation models - particularly if transient - are

empirically tested by plotting the predicted values against the measured results. In this case, a graphical assessment of the model's predictions is carried out. In other cases, numerical methods are used, such as the root mean square error[7], which gives an estimate of the difference between the actual observations and the response predicted by the model. Although a numerical approach is useful in that it provides with a single measurement of the model's performance, the graphical approach can highlight different issues, such as whether the model is constantly over or under-predicting, or if unusual results occur under specific circumstances.

If a model's predictions differ from the empirical results in some way, this may be due, for example, to errors/uncertainties in the model's input variables and/or to errors/uncertainties in the measurements themselves. Sensitivity analysis technique are utilised to assess: a) individual sensitivities, which describe the influence on predictions of variations in each individual input; b) total sensitivities, due to uncertainties in all the input data (Lomas and Eppel, 1992). Individual sensitivities are useful in order to identify the input parameters whose values have to be chosen with particular care, since the model is most sensitive to them. Furthermore, individual sensitivities can help identifying those parameters that have the largest effect on the output variable, which is useful for design purposes, scenarios modelling etc. The total uncertainty in outputs due to *all* the input variables allows the assessment of the maximum accuracy of the model, which is useful in empirical validation studies. Lomas and Eppel (1992) compared three sensitivity analysis techniques, by using three detailed finite difference building simulation programs (ESP, HTB2 and SERI-RES). They examined the Differential Sensitivity Analysis (DSA), the Monte Carlo Analysis (MCA) and the Stochastic Sensitivity Analysis (SSA).

The Differential Sensitivity Analysis (DSA) is generally utilised to assess the individual sensitivities. DSA involves varying one input at a time, whilst the remaining inputs stay fixed at base case value. The changes from the base case in the predicted parameter (Δp_i) are therefore a direct measure of the effect of changes in the input variable *i*. This procedure is repeated for each input

[7] $MeanRootSquareError = \sqrt{\frac{\sum_{i=1}^{n} (predicted_i - measured_i)^2}{n}}$

parameter. It is often assumed that each input is distributed normally about the modal (or average), which is taken as the base-case value. The ratio between an average value for the sensitivity (Δp_i) and the likely range of input parameter change (Δ_i, usually 2.33*standard deviations) is usually the most relevant measure. Provided that all input parameters are varied by the same amount (e.g. 2.33 standard deviation), their total influence on the predicted parameter (Δp_{tot}) can be also estimated, as:

$$\Delta p_{tot} = \sqrt{\sum_i \Delta p_i^2} \qquad [2.3.10]$$

The Monte Carlo Analysis (MCA) generates an estimate of the overall uncertainty in the predictions due to all the uncertainties in the input parameters. In order to do so, a probability distribution is firstly assigned to each input parameter. Then for all parameters, values from within their probability distribution are randomly selected and a simulation undertaken. Simulations are undertaken repeatedly with new randomly-selected values. The total uncertainty in the predictions may be expressed in terms of standard deviation s:

$$s = \sqrt{\frac{1}{N-1}\left(\sum_{n=1}^{N} p_n^2 - N\bar{p}^2\right)} \qquad [2.3.11]$$

where N is the total number of simulation, n is the simulation number and $\bar{p}$ is the mean value of the output parameter. The accuracy of this depends on the number of simulations undertaken and not – as in the DSA case – on the number of uncertain input parameters. However, only marginal improvements in the accuracy are obtained after 60-80 runs. Since all the inputs are perturbed simultaneously, MCA fully accounts for any interactions between inputs, which is not the case for the DSA.

The Stochastic Sensitivity Analysis (SSA) seeks to generate the sensitivity of predictions to the individual parameter uncertainties. In SSA, all the uncertain input parameters are varied simultaneously as the simulation progresses, typically at every time-step. This is in contrast with the other 2 techniques, in which the uncertain parameter(s) are varied before the simulation starts and are then held constant for the duration of the simulation. Although SSA produces both individual and total uncertainties, as well as accuracy estimates of the

uncertainties, SSA is mathematically and computationally far more complex than the other two methods. Furthermore, access to the program code is usually required.

Since DSA produces both individual and total sensitivities, Lomas and Eppel conclude that this can be considered the preferred technique, provided that the models can be assumed to work as linear and superposable systems, with normally distributed input parameters. It should be mentioned that the sensitivity tests discussed in this section require the knowledge of the ranges and/or probability distributions of the input parameters. However, in the previous section the lack of knowledge on bed material properties was highlighted.

Other sources of model's uncertainty might be due to factors such as: abstraction/simplification of phenomena or geometries utilised in the model; solution methods (e.g. explicit vs. implicit method). However, these possible sources of uncertainty are often outside the control of the user.

2.4 The "allergy epidemic" and the role of house dust mites in allergic diseases

In this section the role of house dust mites in health and in the "allergy epidemic" is discussed, in order to highlight the reasons why research in the control of house dust mite infestations is important. A particular focus is given to dust mites and asthma, since asthma is arguably the most debilitating allergic disease, with potential fatal consequences. This topic is particularly relevant for the UK, where asthma prevalence is higher than in other westernised countries (Masoli *et al.*, 2004). Building scientists have developed an interest in HDM research, particularly in the past two decades - due to the rising importance of asthma, and to the role of house dust mites in allergic diseases. HDM research has become particularly relevant to building science when some authors advocated that the rise in asthma prevalence might be due, at least in part, to greater exposure to dust mite allergens, because of higher moisture levels produced by low ventilation rates obtained as a result of energy efficiency concerns (Howieson *et al.*, 2003).

This section is divided into 3 parts:

1. **Background information** (section 2.4.1): it provides background information and figures on the prevalence of asthma and other allergic diseases, including their impact on public health. The "epidemic" of allergy and asthma is briefly illustrated, as well as research on worldwide variations in asthma prevalence.
2. **Hypothesis on the "allergic epidemic"** (section 2.4.2): it discusses the current hypothesis on the "allergic epidemic" causes, including the role of increased exposures due to greater airtightness levels.
3. **The role of HDM in asthma** (section 2.4.3): this section discusses: a) To what extent HDM exposure "causes" sensitisation and asthma, with an aim to identify the potential for reducing adverse health outcomes in the UK, through the control of HDM infestations; b) Research on the dose-response relationships between HDM exposure and sensitisation/asthma, with an aim to identify threshold levels for HDM exposure. These issues are particularly important when considering the potential for reducing adverse health

outcomes through the psychrometric control of house dust mites in UK housing.

Considering that the models tested in this thesis do not predict the direct impact of HDM allergens on respiratory health, this section might be considered by some readers a little long. However, this section aims to highlight the complexities associated with any study/method attempting to reduce asthma symptoms or to prevent HDM sensitisation and/or asthma onset. This section partly explain why controlled intervention studies on the clinical efficacy of psychrometric control measures are very rarely conclusive.

2.4.1 The "epidemic" of asthma and allergy, worldwide variations

It has been estimated that as many as 300 million people could suffer from asthma worldwide (Masoli *et al.*, 2004), with 5.2 million asthmatics living in the UK (Asthma UK, 2004). Asthma is a chronic inflammatory disease of the airways, which can lead to serious health outcomes - sometimes fatal. It is estimated that asthma accounts for about 1 in every 250 deaths worldwide and that the number of disability-adjusted life years (DALYs) lost due to asthma is similar to that for diabetes or cirrhosis of the liver (Masoli *et al.*, 2004). In the UK one person dies from asthma every 7 hours on average and asthma accounts for 12.7 million work-days lost each year (Asthma UK, 2004). Asthma has been reported as costing the UK £2 billion a year (Chaytor, 2003).

Although a lot is known about asthma, there is some uncertainty on the causes of asthma onset, and even on the definition of asthma itself. At present there is no universally accepted definition of asthma, nor a single test exists which can diagnose the disease. The *Committee on the Assessment of Asthma and Indoor Air* from the US Institute of Medicine (National Academy of Sciences, 2000) defines asthma as a chronic disease of the airways characterised by an inflammatory response involving many types of cells. The intensity of the inflammation is related to the severity of respiratory symptoms and the degree of bronchial hyperresponsiveness. The latter is an abnormal response of the lungs to the inhalation of minor irritants, such as cold air. The inflammatory response may vary from patient to patient, and the symptoms - often episodic - include

wheezing, breathlessness, chest tightness and coughing. Both genetic and environmental factors play important roles in the onset and continuation of the airways inflammation. It is estimated that the genetic factor explains 30-80% of the asthma risk. For the majority of subjects, asthma onset occurs before puberty; however, in some cases children diagnosed with asthma "grow out of asthma" (remission). Asthma remission may be partly due to the difficulties in defining and diagnosing asthma (National Academy of Sciences, 2000).

Asthma occurs in two different forms: allergic asthma (often referred to as *atopic* or *extrinsic*) and non-allergic asthma (often referred to as *non-atopic* or *intrinsic*). Atopy can be broadly defined as the tendency to generate an immunoglobulin E (IgE) response to specific allergens, or a tendency to generate a wheel of greater than 3 mm in response to skin prick testing (Carroll *et al.*, 2006). Allergic sensitisation can be asymptomatic. However, in atopic asthmatics, inhalation of allergens initiates an inflammatory response leading to airways hyperreactivity and asthma symptoms. In non-atopic asthmatics, the symptoms are similar, but the allergic response defined by the presence of IgE antibodies for specific allergens does not occur. Other atopic conditions include eczema, hay fever and rhinoconjunctivities. (National Academy of Sciences, 2000). The relationship between asthma and atopy is discussed further in the next sections.

Two major worldwide studies have contributed towards the worldwide comparison of the prevalence of allergies, asthma and respiratory health: the European Community Respiratory Health Survey (ECRHS: Janson *et al.*, 2001) for adults (aged 20-44) and the International Study of Asthma and Allergies in Childhood (ISAAC: ISAAC Steering Committee, 1998) for children (2 age groups: 6-7 and 13-14). Figure 2.4.1 shows the estimated worldwide prevalence of clinical asthma, which has been considered as equivalent to 50% of the prevalence of "current wheezing" in 13-14 years old children from the ISAAC study (Masoli *et al.*, 2004). The figure shows that the UK is amongst those countries with the highest asthma prevalence.

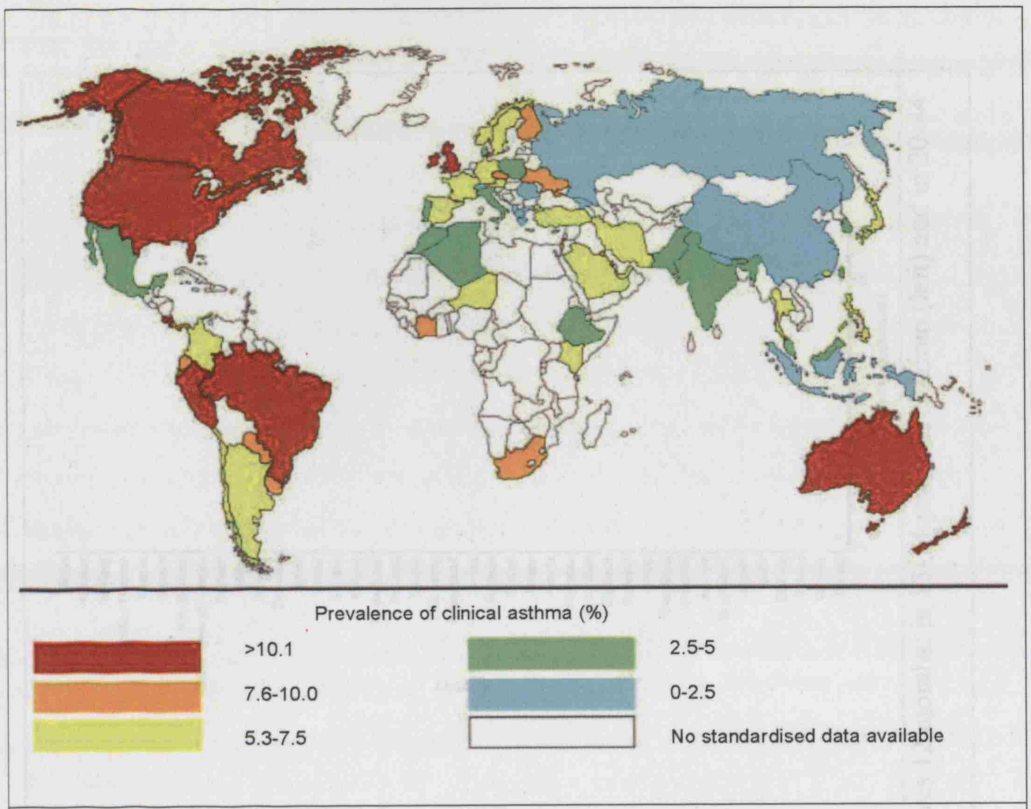

Fig 2.4.1 Worldwide prevalence of clinical asthma (Masoli *et al.*, 2004).

Figure 2.4.2 and 2.4.3 also show that Scotland has the highest prevalence of current asthma symptoms in 13-14 years old children, while Wales has the highest prevalence for 20-44 years old adults. English-speaking countries have one of the highest prevalences, which may be due to genetic and/or cultural factors.

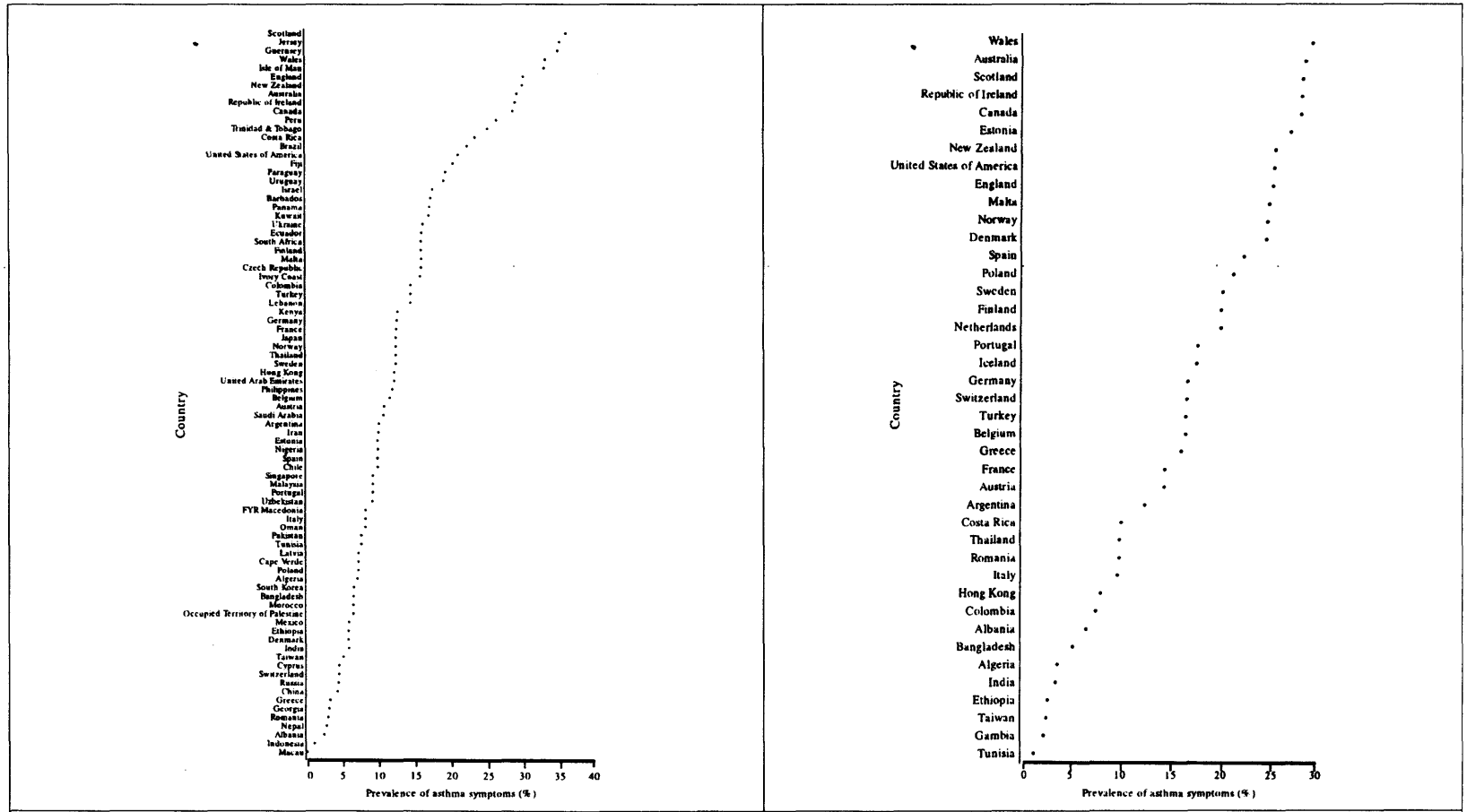

Fig 2.4.2 and **2.4.3** Prevalence of self-reported wheezing in the previous 12 months: in 13-14 year-old children (left) and in 20-44 year-old adults (Masoli *et al.*, 2004)

There are significant geographic variations in asthma prevalence, but the strength of the association between asthma and atopy does not appear to vary significantly worldwide in adults. However, the adult population fraction of asthma attributable to dust mite sensitisation[8] does appear to vary significantly worldwide (Table 2.4.1, section 2.4.3), suggesting that the prevalence of sensitisation to dust mites (and exposure to dust mite allergen) is the main determinant of geographic variations in the population fraction of asthma attributable to atopy (Sunyer *et al.*, 2004). The statistically significant worldwide variations in the risk of asthma attributable to HDM are likely to reflect, at least in part, differences in climatic conditions which in turn can affect dust mite infestations. Indeed, studies have shown that in high altitudes - where the air is drier – mite numbers and asthma cases are low (Charpin, 1988). An analysis of the ISAAC study also concluded that climate may affect the prevalence of asthma and atopic eczema in children. In particular, in Western Europe (57 centres in 12 countries) the prevalence of asthma symptoms increased by 2.7% (95% CI 1.0% to 4.5%) with an increase in the estimated annual mean indoor relative humidity (at 20 °C) of 10% (Weiland *et al.*, 2004). However, a UK-based study (Court *et al.* (2002) revealed that there are no dramatic differences in HDM sensitisation rates across the NHS regions in England (based on data from the 1995-1996 Health Surveys for England). This suggests there might be an insufficient variation in HDM allergen levels (linked to climatic variations) in England.

Several epidemiological studies have reported an increase in the occurrence of atopic diseases over the past 30-40 years, particularly in affluent countries (von Hertzen and Haahtela, 2004). It has been estimated that between the mid-1960s and the mid-1990s there was an increase in asthma prevalence of approximately 5% per year (Jarvis and Burney, 1998). Some authors even refer to an "epidemic" of allergy and asthma (Holgate, 2004; Eder *et al.*, 2006), although others warn that the increase in asthma prevalence may be due, at least in part, to a trend towards applying the "asthma label" to increasingly milder disease (Anderson *et al.*, 2004). Some studies also suggest that this "epidemic" in atopy and asthma may have reached its plateaux in some countries (Fig 2.4.4), including the UK (Anderson *et al.*, 2004; Fleming *et al.*, 2000; Innes Asher *et al.*, 2006).

[8] For a definition of "population fraction of asthma attributable to dust mite sensitisation" see section 2.4.3.

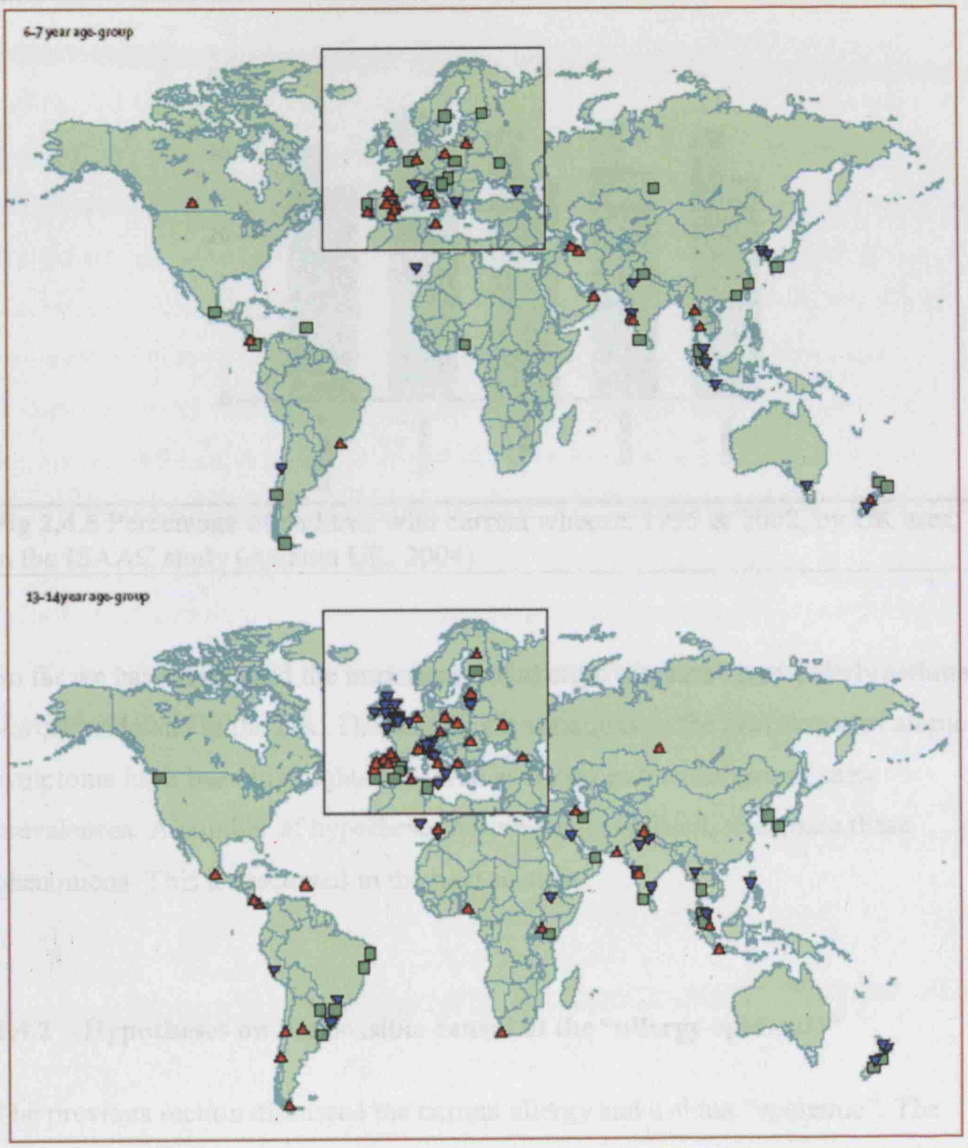

Fig 2.4.4 Changes in the prevalence of asthma symptoms in children, from the ISAAC Study, Phase III (Innes Asher *et al.*, 2006).

The percentage of UK children with current wheeze was 33.9% in 1995, but this figure was reduced to 27.5% in 2002 (Fig 2.4.5). Similar findings have been reported in a Swiss study and in a study based in Rome (Devereux *et al.*, 2003).

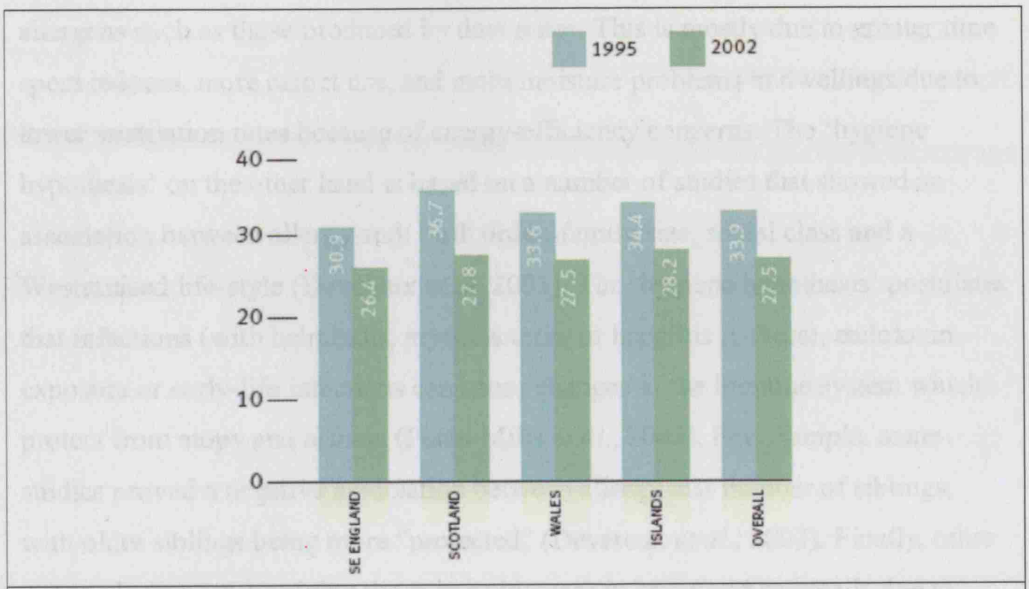

Fig 2.4.5 Percentage of children with current wheeze, 1995 & 2002, by UK area, in the ISAAC study (Asthma UK, 2004).

So far we have discussed the importance of allergic diseases - particularly asthma – worldwide and in the UK. The worldwide variations in the prevalence of atopic symptoms have been highlighted, as well as trends in the changes of such prevalences. A number of hypotheses have been formulated, to explain these phenomena. This is discussed in the next section.

2.4.2 Hypotheses on the possible causes of the "allergy epidemic"

The previous section discussed the current allergy and asthma "epidemic". The worldwide variations in symptom prevalence, which emerged from multi-centre studies, were also highlighted. A number of hypotheses have been formulated, to explain the increase of symptoms prevalence in westernised countries, and the worldwide variations. These hypotheses are discussed in this section.

Several hypotheses have been formulated to explain the increase in allergy and asthma in westernised countries (Fig 2.4.6). The main hypotheses were summarised in a symposium on the increase of allergic disease: a) increased exposure to allergen; b) the 'hygiene hypothesis'; c) changes in diet, and changes in physical activity levels (Devereux *et al.*, 2003). The first hypothesis connects the increase in atopy and asthma to greater exposure and sensitisation to perennial

allergens such as those produced by dust mites. This is mostly due to greater time spent indoors, more carpet use, and more moisture problems in dwellings due to lower ventilation rates because of energy-efficiency concerns. The 'hygiene hypothesis' on the other hand is based on a number of studies that showed an association between allergy and: birth order, family size, social class and a Westernised life-style (Devereux *et al*, 2003). The 'hygiene hypothesis' postulates that infections (with helminths, mycobacteria, or hepatitis A virus), endotoxin exposure or early-life infections can cause changes in the immune system which protect from atopy and asthma (Platts-Mills *et al*., 2005). For example, some studies proved a negative association between allergy and number of siblings, with older siblings being more "protected" (Devereux *et al*., 2003). Finally, other experts have postulated that the current increase in atopy and asthma is due to changes in physical activity levels, increased obesity and changes in diet, with reduced intake of vitamins, minerals and antioxidants (reduced consumption of fresh fruit and vegetables), as well as changes in the types and quantity of lipids.

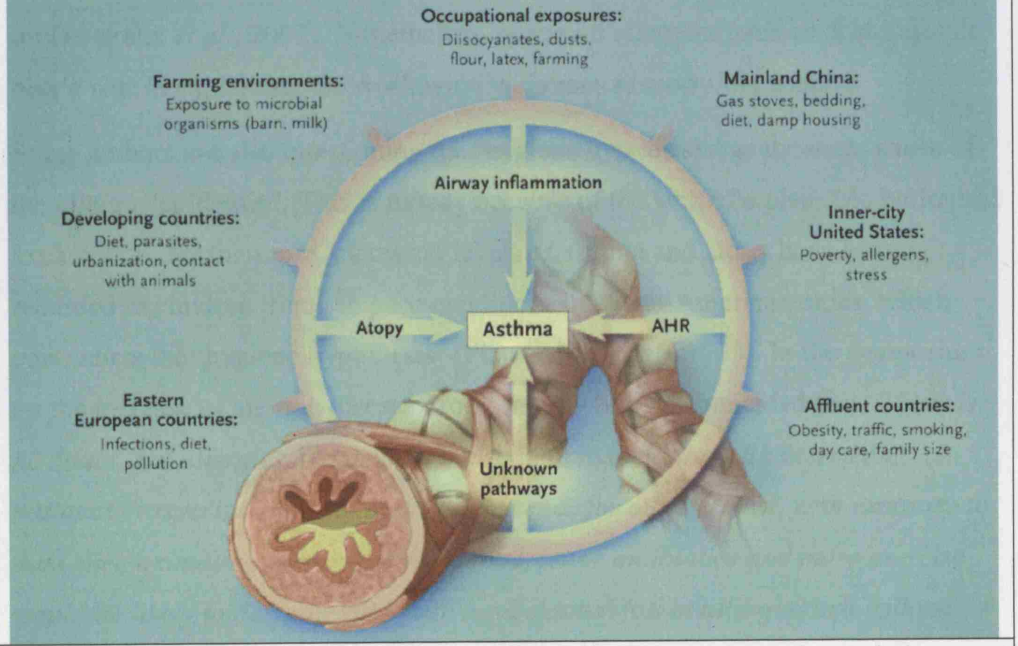

Fig 2.4.6 Possible risk factors for asthma for different settings and populations (Eder *et al*., 2006).

The underlying principles behind these hypotheses is that either people in westernised countries are becoming *more susceptible* to environmental factors, or

they are increasingly *more exposed* to environmental factors (i.e. higher exposure levels). Some authors have advocated that the rise in asthma levels in Westernised countries may be due to recent changes in the building stock, where energy efficiency concerns may have caused excessively low ventilation rates in housing, resulting in high moisture levels which create favourable conditions for house dust mite infestations (Howieson *et al.*, 2003). However, it is extremely difficult to test this hypothesis (see Chapter 1), and most existing data are inadequate for conclusions to be drawn whether ventilation rates *directly* cause ill-health (Davies *et el.*, 2004). Although exposure to perennial allergens is a risk factor for asthma, several experts have concluded that increased exposure to indoor allergens (e.g. dust mites) is unlikely to be the *sole* cause of the atopy and asthma "epidemic" (Holgate, 2004; Platts-Mills *et al.*, 2005). For example, Platts-Mills argues that this epidemic has occurred even in countries such as Sweden, where cat-sensitisation is dominant, as opposed to dust mite sensitisation. It is unlikely that cat ownership has dramatically increased over the past decades, and in Sweden 80% of cat-sensitised children have never lived in a house with a cat (Platts-Mills, in: Devereux *et al.*, 2003). Nonetheless, perennial allergens such as dust mites do play a role in explaining the worldwide variations in atopy and asthma.

Some authors are also questioning the 'hygiene hypothesis' as the *main* cause of the allergy "epidemic". This is mostly because of the lack of a plausible biological explanation. Furthermore, increased levels of asthma and atopy have been recorded in children living in poor conditions in North-American cities, which contradicts the 'hygiene hypothesis' (Platts-Mills *et al.*, 2005). In the symposium on the increase of allergic disease Prof Anthony Seaton concluded that "*There is no doubt that allergies and asthma increase in association with increasing national prosperity. A more 'natural' existence for our children, with exposure to dust, dirt, animals, unprocessed fresh food, fewer antibiotics and more exercise would be likely to be associated with a substantial fall in allergies and asthma*" (Seaton, in: Devereux *et al.*, 2003). It should also be pointed out that environmental factors may be more important in the development of atopy and allergic rhinitis than in the development of asthma (von Hertzen *et al.*, 2004).

In summary, the previous section highlighted that asthma represents a significant burden to society worldwide, with evidence of a potential increase in asthma

symptoms, particularly in westernised countries. Furthermore, studies based on worldwide centres have highlighted that some countries, including the UK, have far higher prevalences of asthma symptoms than others. A number of hypotheses have been formulated to explain this "epidemic" of asthma and allergies. At present, no single hypothesis can adequately explain such an "epidemic", although a combination of lifestyle factors ("cleanliness", diet, physical activity) are all likely to play a role. Increased exposure to perennial allergens is unlikely to have *caused* the "epidemic" on its own, but it does play a role, particularly in the worldwide differences in asthma and atopy. The next section discusses the role of atopy in asthma, with a focus on house dust mite allergens exposure.

2.4.3 Asthma, atopy and house dust mites

As previously illustrated, asthma is the most serious of allergic diseases, being disabling and occasionally fatal. This section discusses to what extent HDM exposure "causes" sensitisation and asthma, with an aim to identify the potential for reducing adverse health outcomes in the UK, through the control of HDM infestations. This section also summarises the results from a number of research studies on the dose-response relationships between HDM exposure and sensitisation/asthma, with an aim to identify threshold levels for HDM exposure. This section is a summary of a more detailed review, provided in Appendix A.2.

Many studies – especially cross-sectional - have demonstrated that asthma is strongly associated with atopy, particularly in children (Cole Johnson *et al.*, 2002). Therefore, a theoretical paradigm has often been advocated in which allergen exposure produces atopic sensitisation in susceptible individuals, and continued exposure then leads to clinical asthma through the development of airways inflammation, bronchial hyperresponsiveness and reversible airflow obstruction (Pearce *et al.*, 1999). The Committee on the Assessment of Asthma and Indoor Air, from the Institute of Medicine of the National Academy of Sciences (US) concluded that: "*There is sufficient evidence of a causal relationship between HDM allergen exposure and exacerbation of asthmatics specifically sensitized to dust mites. Continual exposure to dust mite allergens is also a contributing cause of chronic bronchial hyperreactivity. There is sufficient*

evidence of a causal relationship between dust mite allergen exposure and the development of asthma in susceptible children" (National Academy of Sciences, 2000). The Committee specifies that 'causality' is not intended it in its old-fashioned concept of sufficient *and* necessary cause[9]. Therefore, causality occurs if there is at least one person whose asthma was caused by a certain factor X.

However, there is currently some controversy on the relationships between exposure to aeroallergens (e.g. dust mites), sensitisation (i.e. atopy), and asthma development. One of the issues under discussion is not necessarily whether exposure to dust mite allergen can "cause" asthma in at least one person, but rather whether HDM exposure causes asthma for a *significant* proportion in the asthmatics population. In order to examine this issue, some studies have calculated the population attributable fraction for atopy and asthma. The population Attributable Fraction (AF) can be defined as the proportion of disease cases over a specified time that would be prevented following the elimination of the exposures, assuming the exposures are causal (Rockhill *et al.*, 1998). If exposure (atopy in this case) has an odds ratio for asthma of R, the proportion of exposed cases (i.e. atopic asthmatics) that are attributable to exposure (i.e. atopy) is:

$$AF_{exp}=(R-1)/R \qquad [2.4.1]$$

The proportion of all cases (i.e. asthmatics) in the general population that are attributable to exposure (atopy) is the population attributable fraction, which is:

$$AF_{pop}=P*(R-1)/R \qquad [2.4.2]$$

where P is the proportion of all cases that are exposed (i.e. proportion of atopic asthmatics).

A number of studies found that the population-based of asthma cases in adults attributable to sensitisation to common aero-allergens (e.g. HDM, cat, grass) is approximately 30% (Pearce *et al.*, 1999; Sunyer *et al.*, 2004; Jaakkola *et al.*, 2006). The fraction of asthma attributable to atopy is approximately 60% among atopic cases[10] (Sunyer *et al.*, 2004; Jaakkola *et al.*, 2006). The ECRHS study

[9] *Sufficient* cause: all persons exposed to x will develop asthma; *necessary* cause: all cases of asthma are caused by x

[10] This means that amongst those asthmatics sensitised to common aero-allergens, 60% of asthma cases could theoretically be avoided, if sensitisation were to be removed.

found that the population-based of (prevalent) asthma cases in adults attributable to sensitisation to house dust mites is approximately 18.2% (Table 2.4.1), varying from 19% to 36% in England and Wales (Sunyer *et al.*, 2004). It should be noted that the population fraction of asthma attributable to atopy varies in relation to the definition of asthma and of atopy. If for example atopy is defined in a more stringent way (e.g. four or more positive skin prick tests, rather than just one), the population attributable risk decreases (Pearce *et al.*, 1999). On the other hand, if the definition of asthma is more stringent (e.g. doctor-diagnosed, rather than self-reported wheezing), the AF increases (Sunyer *et al.*, 2004).

Table 2.4.1 (Sunyer *et al.*, 2004). ECRHS (adults, aged 20-44): AF of asthma, defined on the basis of symptoms, caused by specific IgE sensitization and atopy by center

Countries ordered by % of atopy*	Center	HDM	Cat	Tim. grass	Atopy* (95% CI)
Estonia	Tartu	6	17	13	4 (219.0 to 22.0)
Iceland	Reykjavik	35	28	25	40 (22.1 to 64.5)
Spain	Albacete	3	9	7	11 (24.8 to 24.8)
	Oviedo	10	6	15	25 (25.9 to 46.6)
	Galdakao	40	23	13	45 (0.0 to 70.2)
	Huelva	14	22	10	9 (227.3 to 35.5)
	Barcelona (bcn)	32	37	8	61 (227.8 to 88.1)
Norway	Bergen	19	19	18	47 (26.1 to 61.3)
Italy	Pavia	24	0	24	26 (26.1 to 47.8)
	Turin	10	17	20	37 (10.7 to 55.2)
	Verona	21	21	21	44 (6.7 to 66.8)
Sweden	Umea	6	31	26	50 (25.3 to 66.5)
	Goteborg	8	15	13	28 (5.2 to 44.7)
	Uppsala	7	16	20	20 (25.9 to 39.2)
France	Grenoble	12	15	13	16 (216.0 to 39.7)
	Paris	16	18	21	36 (14.4 to 51.6)
	Montpellier	15	11	5	12 (28.8 to 28.3)
	Bordeaux	48	25	23	55 (33.1 to 69.4)
Belgium	South-Antwerp	31	11	27	46 (0.7 to 70.9)
	Antwerp city	37	22	31	55 (17.9 to 75.4)
Germany	Erfurt	214	10	7	11 (226.4 to 37.4)
	Hamburg	19	22	24	43 (19.5, 60.0)
United Kingdom	Cardiff	19	11	11	22 (21.6 to 40.2)
	Ipswich	36	22	23	44 (18.1 to 61.3)
	Norwich	19	20	17	26 (22.2 to 45.9)
	Cambridge	29	12	12	38 (213.8 to 66.6)
The Netherlands	Groningen	54	20	23	58 (13.5 to 79.7)
	Bergen op Zoom	20	15	20	36 (2.1 to 57.7)
	Gellen	19	14	39	26 (217.3 to 53.8)
Ireland	Dublin	35	12	31	26 (29.7 to 50.2)
New Zealand	Hawkes-Bay	14	21	23	14 (229.7 to 43.0)
	Wellington	51	17	18	52 (23.7 to 70.2)
	Christchurch	51	29	30	49 (16.3 to 68.7)
United States	Portland	0	10	29	35 (3.8 to 56.2)
Switzerland	Basel	12	4	17	17 (210.2 to 37.2)
Australia	Melbourne	32	13	25	45 (21.2 to 61.2)
ALL#		18.2 (13.7, 22.4)	14.1 (11.8, 16.3)	17.1 (14.0, 20.1)	30.4 (24.9 to 35.5)
p value for heterogeneity		<.001	.30	.91	.012

*Atopy: IgE sensitization to any of house dust mite, cat, timothy grass, C herbarum, and birch, P judaica, or ragweed.
#AF in the 36 centers estimated with meta-analysis.

The issues discussed so far suggest that if sensitisation to house dust mites could be avoided, this could potentially prevent the occurrence of approximately 19-36% of asthma cases in the general adult population of England and Wales (based on a certain definition of asthma and sensitisation). In order to avoid sensitisation, it would be useful to identify threshold exposure levels below which sensitisation does not occur. In 1989 a team of experts discussed the worldwide problem of dust mite allergens and asthma in an International Workshop under the auspices of the WHO (Platts-Mills and de Weck, 1989). One of the main conclusions of the workshop was the provisional recommendation of threshold levels for mite-allergen exposure. The experts proposed that a level of 2 μg of Der p1 per gram of dust (considered equivalent to 100 mites per gram) should be regarded as a risk factor for sensitisation and the development of asthma. The higher level of 10 μg of Der p1 per gram of dust (considered equivalent to 500 mites per gram) was proposed as a major risk factor for the development of acute asthma in mite-allergic individuals. These conclusions were based on evidence of a linear dose-response relationship between HDM exposure, and sensitisation/asthma development. In 1992, a second International Workshop took place, which confirmed the threshold levels and the dose-response relationship between HDM allergen exposure, HDM sensitisation and asthma development (Platts-Mills *et al.*, 1992). These recommended levels are often referred to in the literature as the "WHO threshold levels" for dust mite allergens and asthma.

However, since the formulation of the "WHO" threshold levels, further research has been carried out, which provides additional insight into the role of HDM allergen exposure on asthma, as well as into the exposure thresholds. Indeed, a *third* International Workshop of experts on dust mites and asthma took place in 1997, highlighting that the pattern of sensitisation to specific allergens reflects the *mean level* of allergen found in the houses of those communities where the patients live. The experts therefore slightly changed their recommendations on threshold levels, concluding that in areas where the *mean level* of dust mite group I allergen in houses is 2 μg of Der p1 per gram of dust or more, sensitisation to mites has consistently been found to be associated with asthma. In the report of the third International Workshop there was no mention of the threshold of 10 μg of Der p1 per gram of dust. This is because the experts concluded that the

relationship between HDM allergen exposure and asthma symptoms is complex, which makes the identification of a threshold level in HDM allergen exposure for asthma exacerbation quite difficult (Platts-Mills *et al.,* 1997).

Since the 1997 WHO workshops on HDM and asthma, a number of studies have further investigated the relationships between HDM exposure and health outcomes. The findings of these studies are discussed in more details in Appendix A.2, while a summary is provided in this section (Sporik *et al.*, 1990; Custovic *et al.*, 1996; Custovic and Chapman, 1998; Marks, 1998; Lau *et al.*, 2000; Cullinan *et al.*, 2004; Cole Johnson et al., 2004; Brussee et al., 2005; Backlund *et al.*, 2006; Carroll *et al.*, 2006; Schram-Bijkerk *et al.*, 2006; Torrent et al., 2006). From a number of studies it can be concluded that there is little doubt that exposure to HDM allergen leads to exacerbation of asthma symptoms in susceptible individuals, and that asthma severity is greater with greater exposure to HDM allergens. However, no single threshold level of HDM allergen exposure can be identified for exacerbation of asthma symptoms. Furthermore, there is some contradictory evidence on the dose-response relationship(s) between exposure to HDM allergens, and HDM sensitisation or asthma onset. This is mostly because these relationships can vary in relation to: study population (i.e. genetic factors and typical exposure levels), family history of asthma/atopy, and other confounding factors – particularly the presence of potentially "protective" factors in the environment, such as endotoxins or extracellular polysaccharides (EPS). Because of these factors, it is unwise to identify a threshold level of HDM exposure for sensitisation or asthma onset, which can be applied to any population. Some evidence suggests that in some study populations HDM sensitisation might occur at intermediate exposure levels, with a bell-shaped relationship, which is possibly also modified by other potentially "protective" factors (Fig 2.4.7). These conclusions should be taken into account in any primary prevention studies aiming at reducing HDM allergen levels for the reduction of HDM sensitisation.

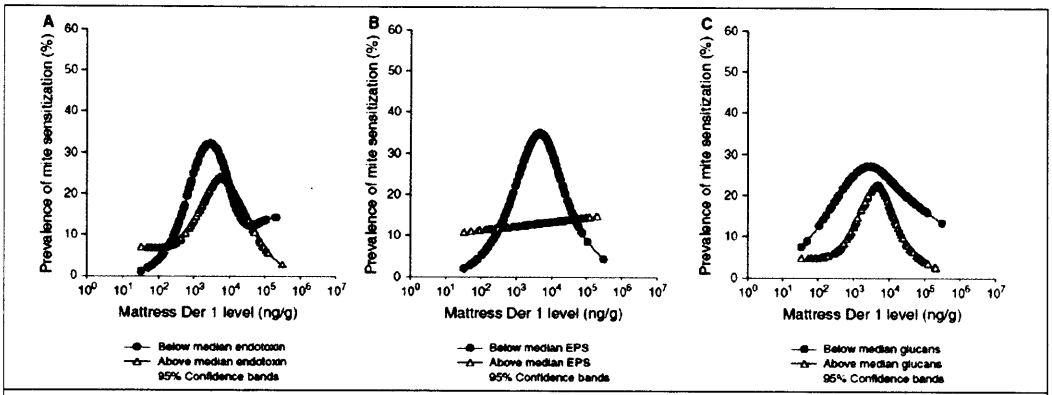

Fig 2.4.7 Smoothed plots of the prevalence of mite sensitisation in relation to mite allergen levels, stratified by: A) endotoxin levels; B) extracellular polysaccharides (EPS) levels; C) glucan levels in mattress dust (Schram-Bijkerk *et al.*, 2006).

The following section is a summary of the literature review.

2.5 Summary of Literature Review

This section summarises the main issues highlighted by the literature review.

House dust mites play an important role in allergic diseases, especially asthma. Asthma is a chronic inflammatory disease of the airways, which can lead to serious health outcomes - sometimes fatal. This disease has been reported as costing the UK £2 billion a year (Chaytor, 2003). A number of studies have shown that there are significant worldwide variations in the prevalence of asthma and other allergic diseases. English-speaking countries (including the UK) have one of the highest prevalences, which may be due to genetic and/or cultural factors. However, some evidence suggests that the prevalence of sensitisation to dust mites (and exposure to dust mite allergen) is the main determinant of geographic variations in the population fraction of asthma attributable to atopy.

Several epidemiological studies have reported an increase in the occurrence of atopic diseases over the past 30-40 years, particularly in affluent countries. Some authors even refer to an "epidemic", although some studies also suggest that this "epidemic" in atopy and asthma may have reached its plateaux in some countries, including the UK. Several hypotheses have been formulated to explain the increase in allergy and asthma in Westernised countries. Some authors have advocated that the rise in asthma levels in Westernised countries may be due to

recent changes in the building stock, where energy efficiency concerns may have caused excessively low ventilation rates in housing, resulting in high moisture levels which create favourable conditions for house dust mite infestations. However, it is extremely difficult to test this hypothesis, and most existing data are inadequate for conclusions to be drawn whether ventilation rates *directly* cause ill-health. Furthermore, although exposure to perennial allergens is a risk factor for asthma, several experts have concluded that increased exposure to indoor allergens (e.g. dust mites) is unlikely to be the *sole* cause of the atopy and asthma "epidemic". At present, no single hypothesis can adequately explain such "epidemic", although a combination of lifestyle factors ("cleanliness", diet, physical activity) are all likely to play a role.

There is overwhelming evidence on the importance of hygrothermal conditions on HDM biology, whereby house dust mites prefer moist and warm environments. By controlling mite microclimates, it is theoretically possible to reduce mite infestations (psychrometric control) and therefore adverse health outcomes. Although several control methods are available for the control of house dust mites, most of these methods are time consuming and therefore they are mostly implemented by those mite-sensitive individuals who are aware of their problem. The psychrometric control method, on the other hand, could be "built-into" housing design or refurbishment - potentially *preventing* adverse health outcomes. However, most experiments on hygrothermal conditions and house dust mites have been carried out under steady-state conditions using laboratory-reared mites. Some studies have shown that wild mites may be more resilient to adverse hygrothermal conditions than laboratory-reared mites. Furthermore, some transient experiments suggest that mites might be able to survive under unfavourable hygrothermal conditions, provided that the indoor RH is favourable for a few hours every day[11]. In addition, most experiments have only addressed changes in one hygrothermal variable (temperature or RH) at a time, whereas this thesis will help demonstrate that both factors are often important. Currently, more research is needed on the impact of transient hygrothermal conditions on house dust mites – preferably 'wild' mites who have not been reared in a laboratory

[11] Nonetheless, in such conditions the growing rate of mite populations and their production of faecal matter are likely to be reduced.

environment for generations, and therefore are potentially less adapted to ideal conditions.

In real dwellings, changes in both temperature and humidity levels will occur on a daily and a seasonal basis, due to the interaction of: weather conditions, building characteristics and occupant behaviour. However, it is difficult to reproduce these realistic transient conditions in a laboratory setting, in order to assess their effect on mites. Therefore, a number of cross-sectional and intervention studies have been carried out, with an aim to: 1) identify those building characteristics which are the most and the least favourable to HDM infestations (cross-sectional studies); 2) prove the efficacy of the psychrometric control approach (intervention studies).

The results from the cross-sectional studies provide some useful indications on the impact of housing characteristics on HDM infestations. For example, there is clear evidence that the age of the sampled object (i.e. mattress, floor, etc.) is a determinant of HDM allergen levels, which is not surprising since HDM allergens are very stable and hence have a long lifetime. The impact of features which affect hygrothermal conditions (e.g. extractor fan, age of the dwelling, etc) is also clear in many studies. There is also some evidence that in the European housing stock – including the UK – adequate ventilation (for example in the form of an extract fan in the kitchen) has the potential of reducing HDM allergen levels. However, in most cases the results from cross-sectional studies cannot be generalised and/or applied to different contexts, since such studies mostly reveal evidence which is only applicable to a specific study population. Furthermore, a number of confounding factors exist in cross-sectional studies on HDM and housing, related to: housing stock characteristics, climatic conditions, occupant habits, etc. Some of these factors are often difficult or expensive to measure (e.g. ventilation rates), which can potentially affect the study outcome.

The clinical efficacy of the psychrometric methods for the control of HDM infestation in temperate climates is also under discussion. Although some intervention studies have yielded some success in reducing mite and allergen levels, others were less successful and no study could prove a statistically significant improvement in asthma symptoms. However, since the principles of psychrometric control are based on sound evidence – i.e. the important role of

hygrothermal conditions on HDM – the lack of evidence of its efficacy is most likely due to a number of issues:

1. Dust mites can survive spells of unfavourable hygrothermal conditions - particularly when these are alternated with brief favourable spells. At present, it is difficult to control such brief spells of elevated RH in dwellings and it is unclear to what extent and how tightly such spells need to be controlled, in order to reduce mite infestation levels. A modelling approach – taking into account all the variables influencing HDM growth - is probably the only way of moving forward in this impasse.

2. Analysis of samples from vacuumed dust currently remains the most used and reliable method for the determination of mite infestation levels. However, this method has a number of limitations. Firstly, mites have sucker-like pulvilli at the ends of their legs, which allow them to cling firmly to their substrate. Consequently, vacuuming only removes a fraction of live mites, which has been estimated as little as 10% of the total population. Furthermore, mites live not only on the surfaces of their habitat but also deep within it. Therefore, vacuuming a mattress, for example, may only give a representative picture of the mite infestation levels of its top layer. In addition, although an attempt has been made for the standardisation of dust sampling, some discrepancies between studies are still likely to occur, for example because of differences in vacuum cleaners (e.g. power), or in dust collectors. It should also be highlighted that HDM allergen levels found in dust are not necessarily representative of exposure levels, which require the allergens to become airborne. Air sampling of HDM allergens is not very common, mostly for the cost and practicalities involved. HDM allergen analysis of settled dust in Petri dishes may add some additional information on HDM allergen exposure profile. The development of new inexpensive methods is needed, for a more reliable assessment of mite infestations - both in terms of allergens and live mites.

3. Mechanical means (e.g. mechanical ventilation) are often considered the ideal psychrometric control methods, since they potentially guarantee a tighter and more restricted control of indoor hygrothermal conditions. However, these methods are expensive; furthermore, they cannot be applied in all

circumstances. For example, mechanical ventilation with heat recovery is more effective in airtight dwellings and involves a large amount of ductwork. Furthermore, the impact of such devices is dependent on the dwelling (e.g. airtightness) and occupant behaviour. Controlling for these factors requires a larger sample size. Consequently, most studies aiming to assess the efficacy of mechanical means often lack the sample size required to detect statistically significant improvements in HDM levels and/or asthma symptoms.

4. Any study aiming to assess the efficacy of psychrometric control methods on HDM infestation levels and on asthma symptoms is faced with a very large number of interrelated factors (see Chapter 1), all of which should ideally be measured at some level. The length of the study can also be crucial. However, this translates in very high costs and most studies had to choose between the number of measured variables and the number of cases under scrutiny. However, so far no "magic formula" for study design has been found. Faster, cheaper and more reliable methods are needed for: assessing levels of mite infestation and of mite allergen exposure, measuring changes in health outcomes, measuring ventilation rates, assessing levels of exposure to other indoor pollutants such as moulds, endotoxins, etc.

5. Many intervention studies on the efficacy of HDM psychrometric control have been carried out by epidemiologists with insufficient knowledge of building physics or by building scientists with insufficient knowledge of HDM biology or asthma. This often resulted in flawed study design and/or data analysis. For example, in some intervention studies it is often assumed that the use of mechanical ventilation will have the same effect on any property. In this case the intervention (e.g. fan) is treated in the same way as a "tablet" is utilised in clinical studies. On the other hand, building scientists may measure the severity of asthma symptoms, without taking into account the impact of medications. However, although a multidisciplinary approach is desirable, this is often hindered by difficulties in obtaining funds - since it becomes unclear which research council or governmental body should fund the research.

Due to the above difficulties in devising and funding intervention studies on HDM psychrometric control, it is perhaps unsurprising that not many of such studies have been carried out in recent years. A modelling approach, on the other hand,

can be very useful in order to disentangle many of the factors which are crucial in any research on the psychrometric control of house dust mites. However, at present there are very few models which can simulate the effect of room conditions on beds, and the impact of bed hygrothermal conditions on mite populations. In particular, apart from the models tested in this thesis, there are no other models which can predict the effect of *transient* room conditions on a population of mites in a bed, based on *three-dimensional* heat and moisture transfer calculations.

For given boundary conditions, hygrothermal conditions in a mattress are determined by the amount of heat and moisture transfer through the mattress itself. It is possible to establish the temperature and RH distributions within a medium resulting from conditions imposed on its boundaries, by referring to the heat and water vapour diffusion differential equations. In order to solve these equations, it is necessary to know a material's: density, thermal conductivity, specific heat capacity, water vapour permeability, and specific moisture capacity. These properties can be measured with different test methods. However, hygrothermal properties can change according to different test methods and ambient hygrothermal conditions. For example, several methods are available to measure the vapour permeability of a material. Some evidence suggests that even using the same method, the water vapour resistance of a material can be identified with a certainty of +/- 10%. It is also important that samples should be tested with the method and conditions that are closer to their end use. If an object is likely to experience a wide range of hygrothermal conditions in its use (e.g. beds), ideally a sample should be tested under most of these conditions. It should be highlighted that the hygrothermal properties of mattresses, beddings and their components are not readily available in published data. It should also be mentioned that compression and geometric deformations usually encountered in mattresses during use add a further problem in the determination of the mattress properties. Furthermore, sprung mattresses comprise a combination of materials, including the metallic springs, and a rather large air gap (compared with air cavities usually encountered in buildings). Convective currents are likely to occur in the mattress's air gap, which are also going to be influenced by the springs. Currently there is no published data on the hygrothermal properties of the air gap in a mattress.

The boundary conditions utilised for modelling hygrothermal conditions in beds have to include room conditions, as well as the effect of heat and moisture outputs from the human body (when the bed is occupied). The human body has a number of mechanisms to maintain its core temperature at approximately 37 °C. These mechanisms include vasodilation, sweating, shivering, changes in metabolic rate, etc. The skin temperature is approximately 34 °C. However, the set-point for the thermoregulation of the human body is not constant, but fluctuates according to endogenous factors, such as: age, vigilance levels, sleep deprivation, thermal adaptation, fever, intake of food or fluids, posture during sleep, heat exposure before sleep, depression, etc. Furthermore, body core and skin temperatures vary according to circadian rhythms and to the sleep phase. Variations of 0.5-2 °C have been observed during sleep in skin temperatures, particularly for the distal skin areas. The rate of evaporation is also affected by sleep phases, as well as by room conditions. Since most studies on sleep and hygrothermal conditions have been carried out under laboratory-controlled conditions, at present it is difficult to estimate the magnitude of variations in heat and moisture outputs *across* individuals, and *within* individuals under realistic ambient conditions. Therefore, monitoring bed hygrothermal conditions in real bedrooms could be useful in this respect.

Finally, this review also considered the methods for testing the validity of models, with a focus on empirical methods. The Differential Sensitivity Analysis was identified as a potentially useful method to be utilised in this thesis, although this method requires the knowledge of the ranges and/or probability distributions of all the input parameters. However, as previously mentioned such knowledge is not completely available for *all* the input parameters required for the models tested in this thesis (e.g. mattress properties).

Once the hygrothermal conditions occurring in a bed have been modelled, it is necessary to model the impact of these conditions on a population of mites. In order to develop such a model, data is required on the impact of combinations of temperature and RH on dust mites. Apart from the two population models tested in this thesis, there is currently only another published HDM population model (Cunningham's model), which is based on data collated from published information on steady-state laboratory experiments on different HDM species.

Therefore, this model is based on data with potentially significant methodological and species-related differences. One of the advantages of the population models tested in this thesis is that they are mostly based on experiments carried out on one mite species (DP), with the same methodology. Furthermore, the Popmite model tested in this thesis is based on experiments on 'wild' DP mites, reared on a 'natural' diet. Details of the models are provided in the following chapter.

The population models currently available - including the ones tested in this thesis) - predict the impact of hygrothermal conditions on mite *populations*. At present, insufficient information is available for the development of a model which also predicts the effect of hygrothermal conditions on *allergen* production. Similarly, there is insufficient evidence to develop a model of the impact of HDM allergen exposure on health. Firstly, although a lot is known about asthma, there is some uncertainty on the causes of asthma onset, and even on the definition of asthma itself. At present there is no universally accepted definition of asthma, nor a single test exists which can diagnose the disease. Secondly, there is currently some controversy on the relationships between exposure to aeroallergens (e.g. dust mites), sensitisation (i.e. atopy), and asthma development. One of the issues under discussion is not necessarily whether exposure to dust mite allergen can "cause" asthma in at least one person, but rather whether HDM exposure causes asthma for a *significant* proportion in the asthmatics population. Some preliminary evidence suggests that if sensitisation to house dust mites could be avoided, this could potentially prevent the occurrence of approximately 19-36% of asthma cases in the general adult population of England and Wales. This figure, however, should be taken with caution: firstly, it is based on asthma prevalence data (i.e. all existing cases), as opposed to incidence data (i.e. new cases only). Secondly, this figure is based on a specific definition of asthma and sensitisation.

In order to develop a model of the impact of HDM allergen exposure on health, it would be useful to identify threshold levels of HDM allergen exposure, below which adverse health outcomes[12] do not occur. There is little doubt that exposure to HDM allergen leads to exacerbation of asthma symptoms in susceptible individuals, and that asthma severity is greater with greater exposure to HDM allergens. However, no single threshold level of HDM allergen exposure can be

[12] Sensitisation, asthma onset, asthma exacerbation.

identified for sensitisation, asthma development or exacerbation, which can be applied to any population. There is some contradictory evidence on the dose-response relationship(s) between exposure to HDM allergens, and HDM sensitisation or asthma onset. This is mostly because these relationships can vary in relation to: study population (i.e. genetic factors and typical exposure levels), family history of asthma/atopy, and other confounding factors – particularly the presence of potentially "protective" factors in the environment, such as endotoxins or extracellular polysaccharides (EPS). Because of these factors, it is unwise to identify a threshold level of HDM exposure, which can be applied to any population. Furthermore, some evidence suggests that in some study populations HDM sensitisation might occur at *intermediate* exposure levels, with a bell-shaped relationship, which is possibly also modified by other potentially "protective" factors. This means that a dwelling with high HDM allergen levels might be potentially less "harmful" than a dwelling with intermediate levels. Therefore, this bell-shaped relationship has a potentially huge impact on any study attempting to prevent the occurrence of sensitisation/asthma through the reduction of HDM allergen levels.

The review of the literature discussed in this chapter highlighted that the psychrometric control of house dust mites is a potentially crucial method for the control of HDM infestations and related adverse health outcomes (e.g. asthma). However, the clinical efficacy of the psychrometric control method has yet to be proven, because of the complex, multidisciplinary nature of this research field. Combined hygrothermal population modelling can play an important role for progress in this field. The models, however, have to be tested in the field, whose complex realistic conditions cannot be easily reproduced in a laboratory setting. Therefore, this thesis aims to cover this knowledge gap, by testing two existing hygrothermal population models against field data, as well as ascertaining their capabilities and the scope for using the models in scenarios modelling.

The following chapter illustrates the models tested in this thesis.

Chapter 2: References

Anderson HR, Ruggles R, Strachan DP, Austin JB, Burr M, Jeffs D, Standring P, Steriu A, Goulding R, (2004), Trends in prevalence of symptoms of asthma, hay fever, and eczema in 12-14 year olds in the British Isles, 1995-2002: questionnaire survey, *BMJ*, 328(7447): 1052-1053.

Arlian LG, (1989), Biology and ecology of house dust mites, Dermatophagoides spp. and Euroglyphus spp., *Immunology and Allergy Clinics of North America*, 9(2): 339-356.

Arlian LG, (1992), Water balance and humidity requirements of house dust mites, *Experimental and Applied Acarology*, 16: 15-35.

Arlian LG, Neal J, Vyszenski-Moher DL, (1999a), Fluctuating Hydrating and Dehydrating Relative Humidities Effects on the Life Cycle of Dermatophagoides farinae (Acari: Pyroglyphidae), *Journal of Medical Entomology*, 36(4): 457-461.

Arlian LG, Neal JS, Vyszenski-Moher DL, (1999b), Reducing relative humidity to control the house dust mite Dermatophagoides farinae, *Journal of Allergy and Clinical Immunology,* 104(4 Pt 1): 852-6.

Arlian LG, Neal JS, Morgan MS, Vyszenski-Moher DL, Rapp CM, Alexander AK, (2001a), Reducing relative humidity is a practical way to control dust mites and their allergens in homes in temperate climates, *Journal of Allergy and Clinical Immunology*, 107(1): 99-104.

Arlian LG, Platts-Mills TA, (2001b), The biology of dust mites and the remediation of mite allergens in allergic disease, *Journal of Allergy and Clinical Immunology*, 107(3 Suppl): S406-13.

Asthma UK, (2004), *Asthma UK Report: Where do we stand? Asthma in the UK today*, http://www.asthma.org.uk/all_about_asthma/publications/where_do_we.html, accessed Dec 2006.

Bach V, Telliez F, Libert JP, (2002), The interaction between sleep and thermoregulation in adults and neonates, *Sleep Medicine Review*, 6(6); 481-492.

Backlund AB, Perzanowski MS, Platts-Mills T, Sandstrom T, Lundback B, Ronmark E, (2006), Asthma during the primary school ages - prevalence, remission and the impact of allergic sensitization, *Allergy*, 61(5): 549-55.

Brussee JE, Smit HA, Van Strien RT, Corver K, Kerkhof M, Wijga AH, Aalberse RC, Postma D, Gerritsen J, Grobbee DE, De Jongste JC, Brunekreef B, (2005), Allergen exposure in infancy and the development of sensitization, wheeze, and asthma at 4 years, *The Journal Of Allergy And Clinical Immunology*, 115 (5): 946-952.

BSI, (1990), *BS EN 7209: 1990. Water vapour permeable apparel fabrics*, London: BSI.

BSI, (2005), *BS 4745: 2005. Determination of the thermal resistance of textiles,* London: BSI.

Carroll WD, Lenney W, Child F, Strange FC, Jones PW, Whyte MK, Primhak RA, and Fryer AA, (2006), Asthma severity and atopy: how clear is the relationship?, *Archives of Disease in Childhood*, 91:405-409.

Chan-Yeung M, Becker A, Lam J, Dimich-Ward H, Ferguson A, Warren P, Simons E, Broder I, Manfreda J, (1995), House dust mite allergen levels in two cities in Canada: effects of season, humidity, city and home characteristics, *Clinical & Experimental Allergy*, 25(3): 240-6.

Charpin D, Kleisbauer JP, Lanteaume A, Razzouk H, Vervloet D, Toumi M, Faraj F, Charpin J, (1988), Asthma and allergy to house-dust mites in populations living in high altitudes, *Chest*, 93 (4): 758-61.

Chaytor Mr, (2003), Oral Answers to Questions, *Commons Hansard Debates*, text for Tuesday 6 May 2003, 404 (389), Column 666.

CIBSE, (1986), *CIBSE Guide, Volume A: Design Data*, London: CIBSE.

Cole Johnson C, Ownby DR, Zoratti EM, Alford SH, Williams KL and Joseph CLM, (2002), Environmental epidemiology of pediatric asthma and allergy, *Epidemiologic Reviews*, 24: 154-175.

Cole Johnson C, Ownby RD, Havstad SL, Peterson EL, (2004), Family history, dust mite exposure in early childhood, and risk for pediatric atopy and asthma, *Journal of Allergy and Clinical Immunology*, 114(1): 105-110.

Colloff MJ, (1987), Effects of temperature and relative humidity on development times and mortality of eggs from laboratory and wild populations of European house dust mites *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 3: 279-289.

Colloff MJ, (1991), Practical and theoretical aspects of the ecology of house dust mites (Acari: Pyroglyphidae) in relation to the study of mite-mediated allergy, *Review of medical and veterinary entomology*, 79(11/12): 611-630.

Couper D, Ponsonby AL, Dwyer T, (1998), Determinants of dust mite allergen concentrations in infant bedrooms in Tasmania, *Clinical and Experimental Allergy*, 28(6): 715-23.

Court CS, Cook DG, Strachan DP, (2002), The descriptive epidemiology of house dust mite-specific and total immunoglobin E in England using a nationally representative sample, *Clinical and Experimental Allergy*, 32(7): 1033-41.

Crane J, Ellis I, Siebers R, Grimmet D, Lewis S, Fitzharris P, (1998), A pilot study of the effect of mechanical ventilation and heat exchange on house-dust mites and Der p 1 in New Zealand homes, *Allergy*, 53(8): 755-62.

Crawley, (2001), Testing and validation of a new building energy simulation program, in: *7th International IBPSA Conference, Rio De Janeiro, Brazil.* Rio De Janeiro: IBPSA.

Crowther D, Horwood J, Baker N, Thomson D, Pretlove S, Ridley I, Oreszczyn T, (2000), *House dust mites and the built environment: a literature review*, http://www.ucl.ac.uk/bartlett-housedustmites/Publications/Publications/review10Oct02.pdf, accessed: Jan 2007.

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

Crowther D and Wilkinson T, (accepted for publication), House Dust Mites, in: Sweeney K and Kampen H (ed.), *WHO Report on Urban Pests and Health*, Bonn: World Health Organization Regional Office for Europe.

Cullinan P, MacNeill SJ, Harris JM, Moffat S, White C, Mills P and Newman Taylor AJ, (2004), Early allergen exposure, skin prick responses, and atopic wheeze at age 5 in English children: a cohort study, *Thorax*, 59:855-861.

Cunningham MJ, (1996), Controlling Dust Mites Psychrometrically - a Review for Building Scientists and Engineers, *Indoor Air*, 6: 249 - 258.

Cunningham MJ, (1998), Direct measurement of temperature and humidity in the microhabitats of house dust mites, *Clinical and Experimental Allergy*, 28: 1104-1112.

Cunningham MJ, (2000), A proposed experimental programme towards control of dust-mites by microclimate modification, in: Siebers R, Cunningham M, Fitzharris P, Crane J, *Proceedings of Mites, Asthma and Domestic Design, III,* Wellington Asthma Research Group, Department of Medicine, Otago University.

Cunningham MJ, Roos C, Gu L, Spolek G, (2004), Predicting psychrometric conditions in biocontaminant microenvironments with a microclimate heat and moisture transfer model - description and field comparison, *Indoor Air*, 14 (4): 235-242.

Custovic A, Taggart SC, Francis HC, Chapman MD, Woodcock A, (1996), Exposure to house dust mite allergens and the clinical activity of asthma, *Journal of Allergy and Clinical Immunology*, 98(1): 64-72.

Custovic A and Chapman M, (1998), Risk levels for mite allergens. Are they meaningful?, *Allergy*, 53(Suppl. 48):71-76.

Davies M, Ucci M, McCarthy M, Oreszczyn T, Ridley I, Mumovic D, Singh J, Pretlove S, (2004), A Review of Evidence Linking Ventilation Rates in Dwellings and respiratory Health – A Focus on House Dust Mites and Mould, *The International Journal of Ventilation*, 3(2): 155-168.

De Boer R, Kuller K, Kahl O, (1998), Water balance of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) maintained by brief daily spells of elevated air humidity, *Journal of Medical Entomology*, 35(6): 905-10.

Devereux G, Barker RN, Burney G, Hardiman M, Holt PG, O'Donnell M, Platts-Mills TA, Seaton A, Strachan DP, Weiss ST, Woodcock A, (2003), The

increase in allergic disease: environment and susceptibility. Proceedings of a Symposium held at the Royal Society of Edinburgh, 4th June 2002, *Clinical and Experimental Allergy*, 33: 394-406.

Dharmage S, Bailey M, Raven J, Cheng A, Rolland J, Thien F, Forbes A, Abramson M, Walters EH, (1999), Residential characteristics influence Der p 1 levels in homes in Melbourne, Australia, *Clinical & Experimental Allergy*, 29(4): 461-9.

Eastop TD and Watson WE, (1992), *Mechanical services for buildings*, Harlow: Longman Scientific & Technical.

Eder W, Ege MJ, and von Mutius E, (2006), The asthma epidemic, *New England Journal of Medicine*, 355(21): 2226 – 2235.

Emenius G, Egmar A, Wickman M, (1998), Mechanical ventilation protects one-storey single-dwelling houses against increased air humidity, domestic mite allergens and indoor pollutants in a cold climatic region, *Clinical and Experimental Allergy*, 28(11): 1389-1396.

Fanger PO, (1970), *Thermal comfort: analysis and applications in environmental engineering*, New York: McGraw-Hill.

Fleming DM, Sunderland R, Cross KW, Ross AM, (2000), Declining incidence of episodes of asthma: a study of trends in new episodes presenting to general practitioners in the period 1989-98, *Thorax*, 55(8):657-61.

Fletcher AM, Pickering CA, Custovic A, Simpson J, Kennaugh J, Woodcock A, (1996), Reduction in humidity as a method of controlling mites and mite allergens: the use of mechanical ventilation in British domestic dwellings, *Clinical and Experimental Allergy*, 26(9): 1051-1056.

Glass EV, Yoder JA, Needham GR, (1998), Clustering reduces water loss by adult American house dust mites *Dermatophagoides farinae* (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 22: 31-37.

Gore RB, Curbishley L, Truman N, Hadley E, Woodcock A, Langley SJ, Custovic A, (2006), Intranasal air sampling in homes: relationships among reservoir allergen concentrations and asthma severity, *Journal of Allergy and Clinical Immunology*, 117(3): 649-55.

Gøtzsche PC, Johansen HK, Schmidt LM, Burr ML, (2006), House dust mite control measures for asthma. *The Cochrane Library*, 3: 1-38.

Hart BJ, (1998), Life cycle and reproduction of house dust mites: environmental factors influencing mite populations, *Allergy*, 53 (Suppl. 48): 13-17.

Hart BJ, Crowther D, Wilkinson T, Biddulph P, Ucci M, Pretlove S, Ridley I, Oreszczyn T, (2007), Reproduction and development of laboratory and wild house dust mites (Acari: Pyroglyphidae) and their relationship to the natural dust ecosystem, *Journal of Medical Entomology,* 44(4): 568-574.

Harving H, Korsgaard J, Dahl R, (1993), House-dust mites and associated environmental conditions in Danish homes, *Allergy*, 48(2): 106-9.

Harving H, Korsgaard J, Dahl R, (1994a), House-dust mite exposure reduction in specially designed, mechanically ventilated "healthy" homes, *Allergy*, 49(9): 713-718.

Harving H, Korsgaard J, Dahl R, (1994b), Clinical efficacy of reduction in house-dust mite exposure in specially designed, mechanically ventilated "healthy" homes, *Allergy*, 49(10): 866-870.

Henane R, Buguet A, Roussel B, Bittel J, (1977), Variations in evaporation and body temperatures during sleep in man, *Journal of Applied Physiology*, 142: 50-55.

Hesselmar B, Aberg B, Eriksson B, Bjorksten B, Aberg N, (2005), Building characteristics affect the risk of allergy development, *Pediatric Allergy and Immunology*, 16(2): 126-31.

Hill, (1998), Quantification of house-dust-mite populations, *Allergy*, 53(48 Suppl): 18-23.

Hirsch T, Range U, Walther KU, Hederer B, Lässig S, Frey G, Leupold W., (1998), Prevalence and determinants of house dust mite allergen in East German homes, *Clinical & Experimental Allergy*, 28: 956-964.

Hirsch T, Hering M, Burkner K, Hirsch D, Leupold W, Kerkmann ML, Kuhlisch E, Jatzwauk L, (2000), House-dust-mite allergen concentrations (Der f 1) and mold spores in apartment bedrooms before and after installation of insulated windows and central heating systems, *Allergy*, 55(1): 79-83.

Holgate ST, (2004), The epidemic of asthma and allergy, *Journal of the Royal Society of Medicine*, 97(3): 103-10.

Howieson SG, Lawson A, McSharry C, Morris G, McKenzie E, Jackson J, (2003), Domestic ventilation rates, indoor humidity and dust mite allergens - Are our homes causing the asthma pandemic?, *Building Services Engineering, Research and Technology*, 24(3): 137-147.

Howieson SG, (2005), *Housing and asthma*, London: Taylor & Francis.

Htut T, Burgess IF, Maunder JW, Basham E, (1996), A pilot study on the effect of one room mechanical ventilation with heat recovery (MVHR) units on house dust mite populations and Der p 1 levels in laboratory simulated bedrooms and on ambient conditions in an occupied bedroom in Cambridge, UK, *International Journal of Environmental Health Research*, 6: 301-313.

Htut T, Higenbottam TW, Gill GW, Darwin R, Anderson PB, Syed N, (2001), Eradication of house dust mite from homes of atopic asthmatic subjects: a double-blind trial, *Journal of Allergy and Clinical Immunology*, 107(1): 55-60.

Hyndman SJ, Vickers LM, Htut T, Maunder JW, Peock A, Higenbottam TW, (2000), A randomized trial of dehumidification in the control of house dust mite, *Clinical and Experimental Allergy*, 30(8): 1172-80.

Incropera FP and DeWitt D, (1996), *Fundamentals of Heat and Mass Transfer*, US: John Wiley & Sons.

Innes Asher M, Montefort S, Björkstén B, Lai C, Strachan DP, Weiland SK, Williams H and the ISAAC Phase Three Study Group, (2006), Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys, *The Lancet*, 368(9537): 733-743.

ISAAC Steering Committee, (1998), Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC, *The Lancet*, 1998, 351(9111): 1225-32.

Jaakkola MS, Ieromnimon A, Jaakkola JJK, (2006), Are atopy and specific IgE to mites and molds important for adult asthma?, *Journal of Allergy and Clinical Immunology*, 117 (3): 642-648.

Janson C, Anto J, Burney P, Chinn S, de Marco R, Heinrich J, Jarvis D, Kuenzli N, Leynaert B, Luczynska C, Neukirch F, Svanes C, Sunyer J, Wjst M; European Community Respiratory Health Survey II, (2001), The European Community Respiratory Health Survey: what are the main results so far? European Community Respiratory Health Survey II, *European Respiratory Journal,* 18(3): 598-611.

Jarvis D, Burney P, (1998), The epidemiology of allergic disease, *BMJ*, 316: 607-610.

Korsgaard J, (1998), Epidemiology of house dust mites, *Allergy*, 53 (Suppl. 48): 36-40.

Kräuchi K, Wirz-Justice, (2001), Circadian clues to sleep onset mechanisms, *Neuropsychopharmacology*, 25(S5): S92-S96.

Kräuchi K, (2006), The human sleep-wake cycle reconsidered from a thermoregulatory point of view, *Physiology and Behaviour*, doi:10.1016/jphysbeh.2006.09.005.

Kumaran, (1996), *IEA Annex 24. Heat, air and moisture transfer in insulated envelope parts. Final report. Volume 3. Task 3: Materials Properties*, Belgium: Laboratorium Bouwfysica.

Lau S, Illi S, Sommerfeld C, Niggemann B, Bergmann R, von Mutius E, Wahn U, and the Multicentre Allergy Study Group, (2000), Early exposure to house-dust mite and cat allergens and development of childhood asthma: a cohort study, *The Lancet*, 356(9239): 1392-1397.

Lomas and Eppel, (1992), Sensitivity analysis techniques for building thermal simulation programs, *Energy and Buildings*, 19: 21-44.

Lowe R, (2000), Psychrometric control of Dust mites in UK Housing, *Building Services Engineering Research and Technology*, 21 (4): 274-276.

Luczynska CM, (1998a), Identification and quantification of mite allergens, *Allergy*, 53(48 Suppl): 54-7.

Luczynska C, Sterne J, Bond J, Azima H, Burney P, (1998), Indoor factors associated with concentrations of house dust mite allergen, Der p 1, in a

random sample of houses in Norwich, UK, *Clinical and Experimental Allergy*, 28(10): 1201-9.

Macdonald IA, Clarke JA, Strachan PA, (1999), Assessing uncertainty in building simulation, in: *Building Simulation 1999 IBPSA Conference, Kyoto, Japan.* Kyoto: IBPSA.

Marks GB, (1998), House dust mite exposure as a risk factor for asthma: benefits of avoidance, *Allergy*, 53(Suppl. 48): 108-114.

Masoli M, Fabian D, Holt S, Beasley R, the Global Initiative for Asthma (GINA), (2004), The global burden of asthma: executive summary of the GINA Dissemination Committee Report, *Allergy*, 59: 469-478.

Matheson MC, Dharmage SC, Forbes AB, Raven JM, Woods RK, Thien FC, Guest DI, Rolland JM, Haydn Walters E, Abramson MJ, (2003), Residential characteristics predict changes in Der p 1, Fel d 1 and ergosterol but not fungi over time, *Clinical and Experimental Allergy*, 33(9): 1281-8.

McCullough EA, Myoungsook K, Shim H, (2003), A comparison of standard methods for measuring water vapour permeability of fabrics, *Measurement Science and Technology,* 14: 1402-108.

McIntyre DA, (1992), The control of house dust mites by ventilation: a pilot study, *Proc. 13th AIVC Conf.,* AIVC, Nice, 497-507.

Mitakakis TZ, Mahmic A, Tovey ER, (2002), Comparison of vacuuming procedures for reservoir dust mite allergen on carpeted floors, *Journal of Allergy and Clinical Immunology*, 109(1): 122-4.

National Academy of Sciences (Committee on the Assessment of Asthma and Indoor Air, Institute of Medicine, U.S.), (2000), *Clearing the Air,* Washington: National Academy Press.

Niven R, Fletcher AM, Pickering AC, Custovic A, Sivour JB, Preece AR, Oldham LA, Francis HC, (1999), Attempting to control mite allergens with mechanical ventilation and dehumidification in British houses, *Journal of Allergy Clinical Immunology*, 103(5 Pt 1): 756-762.

Nordtest, (2003), *Intercomparison on measurement of water vapour permeance, Nordtest project agreement 1529-01. Project report 339-2003*,

http://www.byggforsk.no/visVedlegg.aspx?vedleggID=261&dokumentID=1173, Accessed: Oct 2006.

Okoda S, Suzuki S, Fukui T, Fujiwara Y, Matsuura H, (2005), Basic study for optimal control of in-bed temperature during sleep, in: *Proceedings of the 2005 IEEE Engineering in Medicine and Biology. 27th Annual Conference*, Shanghai: IEEE.

Pearce N, Pekkanen J, and Beasley R, (1999), How much asthma is really attributable to atopy?, *Thorax*, 54: 268–272.

Pearce N, Douwes J, Beasley R., (2000), Is allergen exposure the major primary cause of asthma?, *Thorax*, 55(5): 424-31.

Pike AJ, Cunningham MJ, Lester PJ, (2005), Development of Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) at constant and simultaneously fluctuating temperature and humidity conditions, *Journal Of Medical Entomology*, 42(3): 266-269.

Platts-Mills T and de Weck A, (chairmen), (1989), Dust mite allergens and asthma – a worldwide problem. Report of an International Workshop, *Journal of Allergy and Clinical Immunology*, 83(2, part 1): 416-427.

Platts-Mills TA, Thomas WR, Aalberse RC, Vervloet D, Chapman MD, (1992), Dust mite allergens and asthma: report of a second international workshop, *Journal of Allergy and Clinical Immunology*, 89(5): 1046-60.

Platts-Mills TA, Vervloet D, Thomas WR, Aalberse RC, Chapman MD, (1997), Indoor allergens and asthma: report of the Third International Workshop, *Journal of Allergy and Clinical Immunology*, 100(6 Pt 1): S2-S24.

Platts-Mills TAE, Erwin E, Heymann P, Woodfolk J, (2005), Is the hygiene hypothesis still a viable explanation for the increased prevalence of asthma?, *Allergy*, 60 (Suppl. 79): 25-31.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (*submitted*), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air*.

Rockhill B, Newman B, Weinberg C, (1998), Use and Misuse of Population Attributable Fraction, *American Journal of Public Health*, 88(1): 15-19.

Saville BP, (1999), *Physical testing of textiles*, Cambridge, England: Woodhead Publishing

Schram-Bijkerk D, Doekes G, Boeve M, Douwes J, Riedler J, Üblagger E, von Mutius E, Budde J, Pershagen G, van Hage M, Wickman M, Braun-Fahrländer C, Waser M, Brunekreef B, the PARSIFAL study group, (2006), Nonlinear relations between house dust mite allergen levels and mite sensitization in farm and nonfarm children, *Allergy*, 61(5): 640-647.

Simpson A, Simpson B, Custovic A, Cain G, Craven M, Woodcock A, (2002), Household characteristics and mite allergen levels in Manchester, UK, *Clinical & Experimental Allergy*, 32(10): 1413-9.

Sporik R, Holgate ST, Platts-Mills TA, and Cogswell JJ, (1990), Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. A prospective study, *The New England Journal of Medicine*, 323(9): 502-507.

Sundell J, Wickman M, Pershagen G, Nordvall SL, (1995), Ventilation in homes infested by house-dust mites, *Allergy*, 50(2): 106-12.

Sunyer J, Jarvis D, Pekkanen J, Chinn S, Janson C, Leynaert B, Luczynska C, Garcia-Esteban R, Burney P, Anto JM; European Community Respiratory Health Survey Study Group, (2004), Geographic variations in the effect of atopy on asthma in the European Community Respiratory Health Study, *Journal of Allergy and Clinical Immunology*, 114(5): 1033-1039.

Svennberg K, (2003), *Determination of moisture properties for materials exposed to the indoor air, Report TVBH-3042 Lund*, Lund: Building Physics LTH, Lund University.

Svennberg K, Claesson J, Wadso L, (2006), Microclimates in beds- field measurements and simulation of temperature and moisture conditions in two bed systems, in: Svennberg K, *Moisture buffering in the indoor environment, Report TVBH_1016 Lund*, Lund: Building Physics LTH, Lund University.

Torrent M, Sunyer J, Muñoz L, Cullinan P, Iturriaga MV, Figueroa C, Vall O, Taylor AN and Anto JM, (2006), Early-life domestic aeroallergen exposure and IgE sensitization at age 4 years, *Journal of Allergy and Clinical Immunology*, 118(3): 742-748.

Tovey ER, Mitakakis TZ, Sercombe JK, Vanlaar CH, Marks GB, (2003), Four methods of sampling for dust mite allergen: differences in 'dust', *Allergy*, 58(8): 790-4.

Trechsel HR ed, (1994), *Moisture control in buildings*, Philadelphia: The American Society For Testing and Materials.

van den Bemt L, Vries MP, Knapen L, Jansen M, Goossens M, Muris JWM, Schayck CP, (2006), Influence of mattress characteristics on house dust mite allergen concentration, *Clinical & Experimental Allergy*, 36 (2): 233-237.

van Strien RT, Koopman LP, Kerkhof M, Spithoven J, de Jongste JC, Gerritsen J, Neijens HJ, Aalberse RC, Smit HA, Brunekreef B, (2002), Mite and pet allergen levels in homes of children born to allergic and nonallergic parents: the PIAMA study, *Environmental Health Perspectives*, 110(11): A693-A698.

van Strien RT, Gehring U, Belanger K, Triche E, Gent G, Bracken MB, Leaderer BP, (2004), The influence of air conditioning, humidity, temperature and other household characteristics on mite allergen concentrations in the northeastern United States, *Allergy*, 59(6): 645.

Vihna J, Mikkil A, Valovirta I, Lindberg R, (2002), Water vapour resistance of materials as a function of humidity, *Building Physics 2002- 6th Nordic Symposium*, http://www.ivt.ntnu.no/bat/bm/buildphys/proceedings.php3, Accessed: Oct 2006.

von Hertzen LC, Haahtela T, (2004), Asthma and atopy - the price of affluence?, *Allergy*, 59(2):124-37.

Warner JA, Frederick JM, Bryant TN, Weich C, Raw GJ, Hunter C, Stephen FR, McIntyre DA, Warner JO, (2000), Mechanical ventilation and high-efficiency vacuum cleaning: A combined strategy of mite and mite allergen reduction in the control of mite-sensitive asthma, *Journal of Allergy and Clinical Immunology*, 105(1 Pt 1): 75-82.

Weiland SK, Husing A, Strachan DP, Rzehak, Pearce P, and the ISAAC Phase One Study, (2004), Climate and the prevalence of symptoms of asthma, allergic rhinitis, and atopic eczema in children, *Occupational and Environmental Medicine,* 61: 609-615.

Wickens K, Mason K, Fitzharris P, Siebers R, Hearfield M, Cunningham M, Crane J, (2001), The importance of housing characteristics in determining Der p 1 levels in carpets in New Zealand homes, *Clinical & Experimental Allergy*, 31(6): 827-35.

Wickens K, Lane J, Siebers R, Ingham T, Crane J, (2004), Comparison of two dust collection methods for reservoir indoor allergens and endotoxin on carpets and mattresses, *Indoor Air*, 14(3): 217-22.

Zock JP, Heinrich J, Jarvis D, Verlato G, Norbäck D, Plana E, Sunyer J, Chinn S, Olivieri M, Soon A, Villani S, Ponzio M, Dahlman-Hoglund A, Svanes C, Luczynska C and the Indoor Working Group of the European Community Respiratory Health Survey II, (2006), Distribution and determinants of house dust mite allergens in Europe: The European Community Respiratory Health Survey II, *Journal of Allergy and Clinical Immunology,* 118(3): 682-90.

CHAPTER 3:

DESCRIPTION OF MODELS

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3.1 Introduction

As already illustrated in Chapter 1, this thesis tests 2 combined hygrothermal population models. The first set of model is a "simple" steady-state model, combining the bed model BED with the population model MPI (Figure 1.2.2 in Chapter 1). The second set of models is a "complex" transient 3-dimensional model, which combines the bed model Lectus with the population model Popmite (Figure 1.2.3 in Chapter 1). In this chapter the models tested in this thesis are described, starting with the steady-state model hygrothermal BED (section 3.2), followed by the transient hygrothermal model Lectus (section 3.3), the steady-state population model MPI (section 3.4) and the transient population model Popmite (section 3.5). The chapter ends with a summary conclusion (section 3.6). The models description is largely based on published papers, where further detailed information can be found.

3.2 The BED Model

The BED model is a steady-state one-dimensional hygrothermal bed model, which predicts the average monthly temperature and RH within the "bed core" (defined as the occupied space between the mattress and the covering), given the average monthly temperature and RH of the bedroom (Pretlove *et al.*, 2005). Monitored values can be used for the bedroom conditions, if available. Alternatively, values predicted by models such as the validated Condensation Targeter II could be used (Oreszczyn and Pretlove, 1999).

The BED model assumes that when the bed is unoccupied, the bed core is in equilibrium with room conditions. When the bed is occupied, it is assumed that the bed core is at 34 °C (average skin temperature, in Fanger, 1970) and that the thickness of the bed cover (e.g. duvet) is adjusted in order to maintain this constant temperature. In reality the thickness of the cover on a bed will not vary each month. However, as an occupant begins to feel too warm in bed they may cover less of their body with the cover, or they may move within the bed. This

could be considered equivalent to changes in the thickness of the bed cover, following the formula:

$$d_{cover} = k_{cover}\left[\left(\frac{1}{\frac{2Q_{bed}}{\Delta T} - U_{mattress}}\right) - R_{s.cover}\right] \qquad [3.2.1]$$

where d_{cover}, k_{cover} and $R_{s.cover}$ are the cover's: thickness (m), its thermal conductivity ($Wm^{-1}K^{-1}$) and its surface thermal resistance (m^2KW^{-1}), respectively. $U_{mattress}$ is the mattress thermal transmittance ($Wm^{-2}K^{-1}$), while ΔT is the temperature difference between the core of the bed (34°C) and the room temperature (°C). The sensible metabolic heat gain into the bed (Q_{bed}) is the key variable in equation 3.2.1, and it is calculated as follows:

$$Q_{bed} = M - (R + C + E_{re} + L + E_d) \qquad [3.2.2]$$

where M is the total metabolic heat gain (assumed as 40 W/m^2 of body surface area for sleeping). The total metabolic heat gain (M, in W) is the sum of the sensible metabolic heat gains into the bed (Q_{bed}, in W/m^2), radiant heat losses from the head (R, in W), convective heat losses from the head (C, in W), latent respiration heat losses (E_{re}, in W), dry respiration heat losses (L, in W) and latent heat losses by skin diffusion (E_d, in W). Each of these separate components is determined within the BED model using adapted formulae published by Fanger (1970).

Once the cover thickness is calculated in relation to the thermal comfort requirements, the moisture calculation then uses the varying monthly cover thickness for the calculation of the moisture in the bed. In an occupied bed, the human body uses sweating primarily to regulate temperature, not vapour pressure. Since the BED model assumes an occupied bed core temperature of 34°C, the impact of sweating has been ignored in this simple model and it is assumed that the moisture performance is dominated by vapour diffusion through the body. Therefore, the vapour pressure within the core of the occupied bed (VP_{bed}) is calculated using the saturated vapour pressure at skin temperature (SVP_{skin}), the

vapour pressure of the air in the room (VP_{room}) and the vapour resistance values for: the body (VR_{body}), the mattress ($VR_{mattress}$) and the cover (VR_{cover}), according to the formula:

$$VP_{bed} = \frac{\left(\frac{SVP_{skin}}{VR_{body}} + \frac{VP_{room}}{VR_{mattress}} + \frac{VP_{room}}{VR_{cover}}\right)}{\left(\frac{1}{VR_{body}} + \frac{1}{VR_{mattress}} + \frac{1}{VR_{cover}}\right)} \qquad [3.2.3]$$

Monitoring of beds showed that bed conditions tend to change relatively quickly at the start of human occupation and revert back to room conditions relatively quickly after occupation. Thus, the impact of moisture absorption and desorption will not have a significant effect on the average monthly environmental predictions and so have not been accounted for. Accordingly, when the bed is unoccupied, the vapour pressure within the core of the bed is assumed to be the same as the vapour pressure of the room air ($VP_{bed} = VP_{room}$). Once the occupied and unoccupied vapour pressures have been determined, the relative humidity for the occupied bed (RH_{occ}) is determined using the occupied bed temperature (34°C) and vapour pressure (VP_{bed}), while the relative humidity for the unoccupied bed (RH_{unocc}) is determined using the unoccupied bed temperature ($=T_{room}$) and vapour pressure ($=VP_{room}$). The 24-hour average bed core relative humidity (RH_{bed}) is then determined using the number of hours that the bed is occupied (t_{occ}):

$$RH_{bed} = \frac{(RH_{occ} \cdot t_{occ}) + (RH_{unocc} \cdot (24 - t_{occ}))}{24} \qquad [3.2.4]$$

The thermal calculation for the bed core is simple, since the occupied bed temperature is assumed to be 34°C (the comfort temperature) and the unoccupied bed temperature is assumed to be the ambient room temperature (T_{air}), measured or predicted by the Condensation Targeter II model. Therefore, given the number of hours that the bed is occupied (t_{occ}), the average monthly temperature (T_{bed}) in the bed core is:

$$T_{bed} = \frac{(34 \cdot t_{occ}) + (T_{air} \cdot (24 - t_{occ}))}{24} \qquad [3.2.5]$$

Pretlove *et al.* (2005) also carried out a preliminary validation exercise of the BED model, by comparing its predictions with the average monthly hygrothermal conditions measured in 3 beds (bed cores) over a year. The authors examined the predictions that the BED model provided when using measured bedroom data, as well as the predictions that the model provided when using bedroom conditions as predicted by Condensation Targeter II. Given actual rather than simulated room conditions, the results indicate that the BED model predicts the conditions of temperature and relative humidity within the bed core with a reasonable degree of accuracy, considering the accuracy of the dataloggers used for monitoring. The mean deviation between measured and predicted results in the bed was 0.7°C for the temperature and 4.2% for the RH. However, there was a consistent tendency for BED to slightly under-predict relative humidity. The results for measured and predicted conditions in bedrooms show that Condensation Targeter II also performed well (mean deviation 1.1°C for temperature and 4.5% for relative humidity), although it too slightly under-predicts the average conditions of relative humidity in the bedroom. Pretlove *et al.* note that the reasons for these under-predictions might include assumptions relating to material properties of the bedding, comfort, sweating, and heat and moisture transfer.

3.3 The Lectus Model

The Lectus model is a transient 3-dimensional bed model (Ridley *et al.*, submitted). The model uses as input the hourly temperature and relative humidity of the bedroom, the properties of the mattress materials, and boundary conditions on the surfaces of the mattress. The model splits the bed into a flexible user-defined three-dimensional grid, which can include multiple layers of different materials. Predictions of hourly temperature and relative humidity are made throughout the mattress for a user-defined time step.

The room conditions utilised in Lectus can be either measured values, or predictions from any hygrothermal building simulation programme providing hourly predictions. For the boundary conditions, it is assumed that at all times other than when the bed is occupied and on all surfaces other than the top, the

boundary conditions are the same as the room. When the bed is occupied, the top surface conditions in each zone are assumed to be those illustrated in Figure 3.3.1.

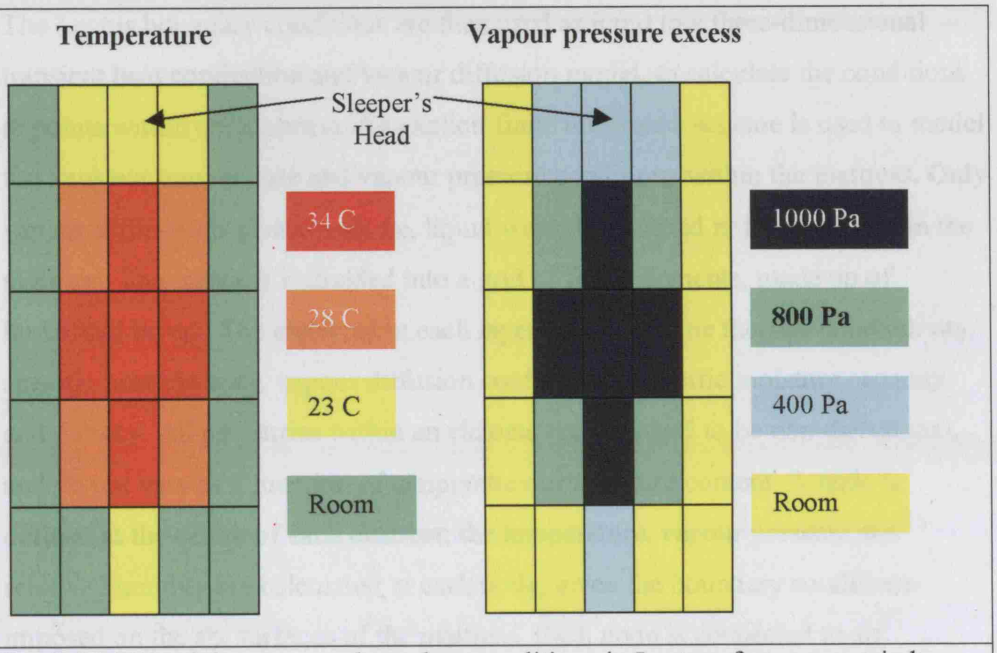

Fig. 3.3.1 Top of the mattress boundary conditions in Lectus, for an occupied sprung mattress.

For example, it is assumed that, when the bed is occupied, on the surface zone corresponding to the chest area the temperature is 34 °C and the Vapour Pressure Excess (VPX) is 1000 Pa. The surface vapour pressure in each zone is then calculated by adding its VPX to the room's vapour pressure. These assumptions were developed by calculating the average conditions measured on the top surface of a laboratory mattress, when the bed was occupied by 10 volunteers (one at a time). Once the occupant leaves the bed, it is assumed that the bed surface conditions gradually go back to equilibrium with the room conditions, following the formula:

$$T\_Bed^{n}=T\_Bed^{n-1}-\{[\Delta T^{n-1}]x[1-(0.5)\hat{}(t/k)]\} \qquad [3.3.1]$$

where T_Bed^{n} is the bed temperature at the time interval n; T_Bed^{n-1} is the bed temperature at the time interval (n-1); ΔT^{n-1} is the temperature difference between the bed and the room, at the time interval (n-1); n is the time interval in minutes; t is the time step in which room conditions are available in minutes (e.g. every 60 minutes); k is a constant, corresponding to the time it takes for the ΔT^{n-1} to be

halved. In Lectus, it is assumed that ΔT^{n-1} is halved every hour (k=60 min). The same formula applies in Lectus for the decay of the vapour pressure.

The Lectus boundary conditions are then used as input to a three-dimensional transient heat conduction and vapour diffusion model, to calculate the conditions at points within the mattress. An explicit finite difference scheme is used to model the transient temperature and vapour pressure conditions within the mattress. Only vapour diffusion is considered, i.e. liquid water is assumed not to form within the mattress. The mattress is divided into a grid of finite elements, made up of horizontal layers. The elements in each layer have the same thermal conductivity, specific heat capacity, vapour diffusion coefficients, specific moisture capacity and density. All properties within an element are assumed to be non-directional, and do not vary as a function of temperature or moisture content. A node is defined at the centre of each element; the temperature, vapour pressure and relative humidity are calculated at each node, given the boundary conditions imposed on the six surfaces of the mattress. Each node is connected to six neighbouring nodes, which may be surface nodes whose values are set by boundary conditions, or other internal nodes within the mattress. Given the boundary conditions and initial conditions at each node, new conditions after the time step $\Delta\tau$ (s), at time $t+\Delta\tau$, may be calculated by applying the Gauss-Seidel iterative method to the explicit finite difference scheme. Hence for temperature, T (°C) for the *ith* node at time p+1:

$$T_i^{p+1} = \frac{\Delta\tau}{C_i}\left[\sum_j \frac{T_j^p - T_i^p}{R_{ij}}\right] + T_i^p \qquad [3.3.2]$$

where , $C_i = \rho_i c_i \Delta Vol_i$, ρ is density ($kg.m^{-3}$), c is specific heat capacity ($J.kg^{-1}.K^{-1}$) and $\Delta Vol = \Delta x \Delta y \Delta z$ is the volume of the element (m^3). R_{ij} describes the thermal resistance between two cells. If those cells are joined together in the z direction then the resistance is: $R_{ij}=R_i + R_j= [dZ_i/(2*dA_{ij}*k_i)]+[dZ_j/(2*dA_{ij}*k_j)]$, where dZ_i is the length of the cell in the z direction, k_i is the thermal conductivity ($Wm^{-1}K^{-1}$) of cell i and dA_{ij} is the area connecting the two cells, equal to dX*dY.

Similarly for vapour pressure V (Pa):

$$V_i^{p+1} = \frac{\Delta\tau}{\xi_i}\left[\sum_j \frac{V_j^p - V_i^p}{Z_{ij}}\right] + V_i^p \qquad [3.3.3]$$

where $\xi_i = \rho_i \varsigma_i \Delta Vol_i$, with ς being the specific moisture capacity ($kg.kg^{-1}.Pa^{-1}$.), and Z_{ij} the vapour diffusion resistance per unit area ($Pa.s.kg^{-1}$) between cell i and j. Again, if those cells are joined together in the z direction, then the resistance is: $Z_{ij}=Z_i + Z_j= [dZ_i/(2*dA_{ij}*\delta_{v\,i})]+[dZ_j/(2*dA_{ij}*\delta_{v\,j})]$, where δ_{vi} is the vapour diffusion coefficient ($kg.m^{-1}.s^{-1}.Pa^{-1}$).

Ridley *et al.* highlight the main advantages of the Lectus model: a) it is three-dimensional instead of one-dimensional; b) the mattress can be made of layers of different materials, and can be described by a very flexible 3D grid, which allows different layers to have a varying number of nodes; c) the mattress is characterised by physical properties which can be directly measured, instead of dimensionless components.

Ridley *et al.* compared the Lectus predictions for the mattress cells (excluding boundary conditions) with the data resulting from the monitoring of a volunteer sleeping in a test bed located in a laboratory chamber. The measured boundary conditions were used for predicting the conditions within the mattress. The sensors (n=75) were located on a 5 by 5 grid on the surface of the mattress and underneath the sheet. At nine locations (arranged in a cross on the surface of the mattress) sensors were additionally placed within the mattress, i.e.1cm from the top, in the middle and 1cm from the bottom of the mattress, and in the duvet. Ridley *et al.* concluded that the model performs acceptably in predicting temperatures and relative humidity to within 1-2 °C and 10% RH. However, the authors also pointed out that they did not have measured material properties for their mattress, so they had to use properties values that achieved the best fit between measured and predicted values.

3.4 The MPI Model

The MPI model predicts the effect of hygrothermal conditions (steady-state) on house dust mite (DP) populations (Crowther *et al.*, 2006). Using a similar approach to Cunningham's model (Cunningham, 2000; see Chapter 2), the MPI output is the mite population index (MPI), such that 1.1 indicates 10% population growth and 0.9 indicates 10% population decline. To provide data for the model, laboratory experiments have been carried out using lab cultures of

Dermatophagoides pteronyssinus (DP). The population change was observed for DP mites held in steady-state conditions at different combinations of temperature and RH over 21 days. From the results, a best-fit equation was derived which forms the basis of the MPI model. The laboratory results also enabled a new term to be defined: the *Population Equilibrium Humidity*, PEH: the RH for a given temperature at which house dust mite populations neither grow nor decline. PEH is similar to Critical Equilibrium Humidity - the RH below which house dust mites are unable to maintain water balance - but relates to a *population* of mites (rather than a physiological phenomenon). The best-fit equation was defined from the experiments:

$$MPI = \exp(a + bY + cY^2 + dX + eXY + fXY^2 + gX^2 + hX^2Y + iX^2Y^2)/100 \quad [3.4.1]$$

where: X = temperature °C, Y = RH% and

a = 2.3397246782 E+01; b = -2.2105777989 E-01; c = -2.4426126335 E-03

d = -1.9681192296 E+00; e = 2.7180575622 E-02; f = 1.8004352184 E-04

g = 3.0361144355 E-02; h = -3.8851175984 E-04; i = -4.1260780086 E-06

The results from the MPI model are broadly similar to those in the Cunningham's model, although in MPI growth peaks more significantly when conditions are ideal and falls off more quickly at extremes of temperature. Crowther *et al.* noted that with more data points it is likely that a sharp fall-off at high RHs would also have been observed.

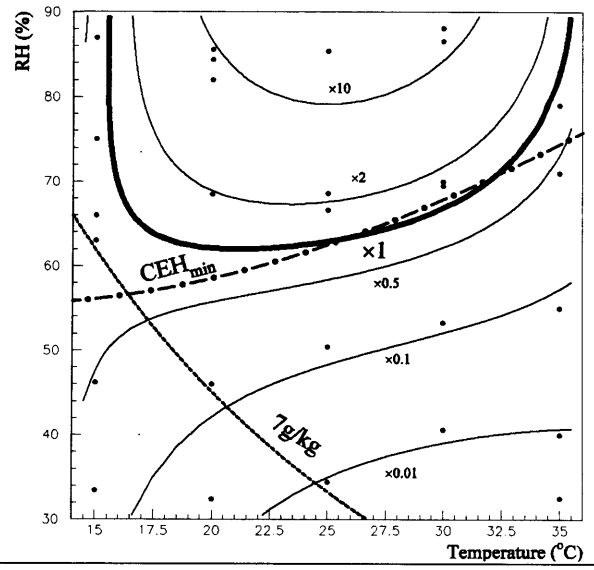

Fig 3.4.1 Plan view representation of MPI lab results with isogrowth lines added (solid line: MPI=1) (Crowther *et al.*, 2006).

Figure 3.4.1 shows isogrowth curves for the lab results, where the solid bold line represents hygrothermal conditions with MPI equals 1 (mite population stable), whilst the other solid curves correspond to MPI values of 2, 0.5 etc. The heavy dashed line is an estimation of the CEH for DP mites, based on published data for DF mites and 4 points for DP. The graph shows that CEH follows closely the MPI=1 curve for mid-range hygrothermal conditions, but diverges sharply at temperatures below 17 °C and above 32 °C. The curve marked on the graph as 7 g/kg represents an absolute humidity value below which it was originally believed mites did not grow, before it was understood that RH is more important than absolute humidity for mites (the Kosgaard limit, see Chapter 2). Therefore, whilst Cunningham had assumed that the CEH for individual mites is equivalent to a *Population* Equilibrium Humidity (PEH), Figure 3.4.1 suggests this may not be the case[1]. A possible reason for this difference is that CEH values have often been estimated by using adult mites, while PEH values represent the net effect of certain hygrothermal conditions on an entire population at all life stages.

[1] Note: the Population Equilibrium Humidity (PEH) corresponds to RH values where the MPI=1.

3.5 The Popmite Model

In this thesis, version 7d of the Popmite model is tested. This is a further development of the version published in Biddulph *et al.* (2007)[2], which forms the basis of version 7d, but was still effectively a steady-state model. In this section, version 7d is described, which is the first population model able to predict the effect of transient (hourly) hygrothermal conditions on a population of DP mites. Since Popmite 7d is based on laboratory experiments utilising 'wild' DP mites feeding on a natural diet of skin and dust (Hart et al., 2007), this population model is potentially better at simulating realistic conditions, than models based on experiments with laboratory-reared mites (e.g. MPI, Cunningham's model).

Both the Cunningham model (Cunningham, 2000) and the MPI model (see previous section) utilise the simplifying assumption that there exists a population growth multiplication factor which is constant at a particular temperature and RH combination, regardless of the structure of the population (i.e. number of eggs, juveniles, adults; development stage, etc.). However, mites at each phase of the life cycle have different development times and mortalities, depending on the hygrothermal conditions, which also affect the fecundity of female adult mites (see Chapter 2). Consequently, certain hygrothermal conditions will have different effects on HDM populations with a different structure. In Popmite it is assumed that a population of mites will consist of batches of individuals at any one of the phases of their life cycle. Each batch will be at a unique development stage within the phase and will be progressing through the phase at a rate which depends on the hygrothermal environment. Hygrothermal conditions will also play a role in determining the chances of survival and the rate at which adult females lay eggs. In particular, the physiological response of mites in a specific phase is determined by:

1. The percentage development rate, which is dependent on the temperature (the warmer it is, the faster the development, for all life stages);
2. The water loss, determined by hygrothermal conditions. In high RH conditions, mites (adults and juveniles) absorb moisture actively from the air and retain a large internal water reserve. In constantly low RH conditions,

[2] See Appendix A.0

mites gradually loose water until their internal water reserve is depleted, eventually dying. If however the RH increases above the Critical Equilibrium Humidity (CEH) before the internal water reserve is completely depleted, mites can recover very rapidly. In Popmite 7d, each mite is assumed to have two "water tanks": a "slow tank", containing the bulk of the water which is slowly lost by diffusion, through the mite's skin. The "slow tank" is replenished with water from the "fast tank", which actively and rapidly takes moisture from the atmosphere when the RH is above CEH. The "fast tank" in Popmite is utilised to replicate the mechanism of moisture absorption from the air via a hygroscopic solution running from the suprocloxal glands along a small channel to the preoral cavity. During periods of low RH the "fast tank" rapidly loses moisture and is sealed by the crystallised salt. Once this has occurred, moisture is only lost via diffusion from the "slow tank". Once the mites have lost a sufficient amount of water, 10% of the mite population dies every hour. For juvenile mites exactly the same mechanism is employed, but the size of the tanks is a fraction of the adult size, depending on the age of the juvenile. The CEH for DP mites which controls this mechanism is temperature-dependent and it is calculated based on Arlian's formulas for DF mites (Arlian and Veselica, 1981), and on observations of mortality rates for DP adults ('wild') in relation to temperature and RH conditions from laboratory experiments by Hart *et al.* (2007). The formula for the CEH in Popmite 7d is:

$$CEH_i = 54.5 - (0.005 - T_i) + \left[525.6/(T_i - 39.3)^2\right] \qquad [3.5.1]$$

where CEH_i is the Critical Equilibrium Humidity at the time step i, corresponding to the temperature T_i.

3. The females will only lay eggs once they have consumed a sufficient amount of food, and have a sufficient water reserve. The food consumption is determined by hygrothermal conditions, whereby the higher the RH, the greatest the food consumption. Since insufficient published information was available on eating rates at different hygrothermal conditions, this parameter had to be adjusted so that Popmite predictions matched extisting laboratory experiments.

All of the above parameters were set in order to reproduce measured fecundity and mortality rates of individual DP wild mites fed on a "natural" diet, which were exposed to constant RH and T combinations in a set of experiments by Hart *et al.* (2007). The parameters were also further modified in order to reproduce the final population sizes of 20 mites held in a range of experiments with transient conditions after 6 weeks. It should be mentioned that Popmite 7d does not include restrictions on mite growth due to food or space availability, due to lack of sufficient information on these parameters. Mite movement/migration is also not currently modelled, for the same reason. A paper on this version of Popmite is currently being prepared by Biddulph *et al.*

Figure 3.5.1 shows a typical output of the Popmite model, with a starting population of 100 eggs kept under certain hygrothermal conditions for 30 days. The plot shows the number of mites as a function of time. The graph shows that for example after 4 days, the eggs turn into juveniles which turn into adults after approximately a further 9 days. At the same point the adults produce almost 200 eggs, which after 4 days turn into juveniles. On the 27th day some of these juveniles turn into adults, which lay some eggs.

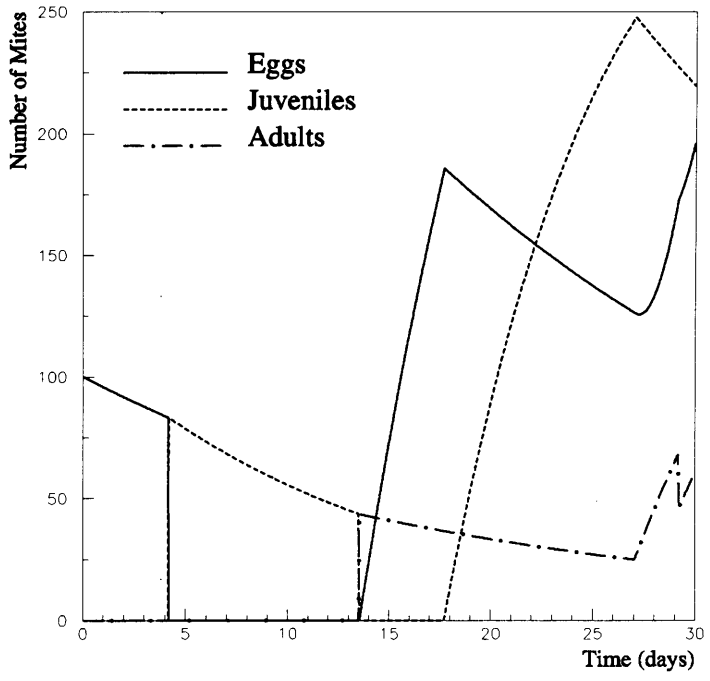

Fig 3.5.1 Typical output from Popmite (Biddulph *et al.*, 2007)

3.6 Summary conclusions

This chapter described the 4 models tested in this thesis. The chapter highlighted that the 2 bed models and the 2 population models have been developed independently, based on different approaches. The complex set of models (Lectus-Popmite) was developed in order to be able to model the impact of transient conditions in a 3-dimensional bed on a mite population. As well as testing the models, this thesis will also examine whether it is necessary to employ such a complex model, or whether in most circumstances the simple model (BED/MPI) can sufficiently predict the impact of changes in building design and occupant behaviour on mite infestations in beds.

Chapter 3: References

Arlian and Veselica, (1981), Effect of temperature on the equilibrium body mass in the mite Dermatophagoides farinae, *Physiological Zoology*, 54(4): 393-399.

Biddulph P, Crowther D, Leung B, Wilkinson T, Hart B, Oreszczyn T, Pretlove S, Ridley I, Ucci M, (2007), Predicting the population dynamics of the house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle, *Experimental and Applied Acarology*, 41(1-2): 61-86.

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

Cunningham MJ, (2000), A proposed experimental programme towards control of dust-mites by microclimate modification, in: Siebers R, Cunningham M, Fitzharris P, Crane J, *Proceedings of Mites, Asthma and Domestic Design, III,* Wellington Asthma Research Group, Department of Medicine, Otago University.

Fanger PO, (1970), *Thermal comfort: analysis and applications in environmental engineering*, New York: McGraw-Hill.

Hart BJ, Crowther D, Wilkinson T, Biddulph P, Ucci M, Pretlove S, Ridley I, Oreszczyn T, (2007), Reproduction and development of laboratory and wild house dust mites (Acari: Pyroglyphidae) and their relationship to the natural dust ecosystem, *Journal of Medical Entomology,* 44(4): 568-574.

Oreszczyn T and Pretlove S E C, (1999), Condensation Targeter II: Modelling surface relative humidity to predict mould growth in dwellings, *Building Services Engineering Research and Technology*, 20 (3) 143 – 153.

Pretlove SEC, Oreszczyn T, Ridley I, Wilkinson T, Crowther D, (2005), A steady-state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements, *Building Services Engineering Research and Technology*, 26(4): 301-314.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (submitted), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air*.

CHAPTER 4:

METHODOLOGY

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4.1 Overview of research design

This thesis aims to test the hypothesis that combined population-hygrothermal model(s) can adequately predict field data and that the model can be a valuable tool for scenarios modelling and intervention studies focused on the psychrometric control of house dust mites. In particular, this thesis aims to:

1. Establish whether the models' predictions are satisfactory, in relation to fieldwork data;
2. Assess the models' capabilities, including the advantages and limitations of the steady state model versus the transient model.
3. Ascertain the scope for using the models in order to establish adequate design and occupant behaviour strategies for the psychrometric control of house dust mites in UK dwellings.

The models validity was tested empirically, through a comparison with fieldwork data. As already mentioned in Chapter 1, the comparison with field data (as opposed to laboratory data) is important for two main reasons:

1. In the literature review, it was highlighted that the set-point for the thermoregulation of the human body is not constant, but fluctuates according to endogenous factors, such as: age, vigilance levels, sleep deprivation, thermal adaptation, fever, intake of food or fluids, posture during sleep, heat exposure before sleep, depression, etc. Therefore, the heat and moisture output from the human body into the bed varies *across* individuals as well as *within* individuals (e.g. food intake etc.). At present, it is unclear to what extent these factors affect the magnitude of changes in heat and moisture output from the human body into the bed. This is mostly because many studies on sleep where carried out in laboratory conditions, under controlled circumstances. Therefore, it is particularly important to test hygrothermal bed models in real settings – rather than simply in laboratory conditions.
2. Most studies examining the impact of hygrothermal conditions on mite populations' growth have been carried out under steady-state conditions. However, beds experience fluctuating conditions, due to room conditions and

to the bed's occupant. The realistic reproduction of fluctuating hygrothermal conditions can be difficult and expensive in a laboratory setting. On the other hand, it is not possible to assess the direct impact of hygrothermal conditions on existing mite populations in real beds, due to difficulties associated with sampling live mites – which for example cling to mattress fibres when an attempt is made to vacuum them. Therefore, it was necessary to develop an innovative technique, in order to be able to study the effect of realistic transient hygrothermal conditions on mite populations.

Given the issues outlined above, the fieldwork study included two main elements:

a) Surveying and monitoring bedroom's and bed's hygrothermal conditions in 25 dwellings. This allowed testing against real cases the bed hygrothermal models, which had previously been tested in a climate chamber (Lectus) and three houses only (BED).

b) Using a novel technique, whereby live DP 'wild' mites were caged with food in a mite and allergen proof 'bag' (similar in size to tea bags) made from porous material, and installed in each monitored bed (see section 4.2.1).

The fieldwork study was carried out in 3 different successive studies: Series 1, Series 2, and Series 3, which are described in more detail in the next section.

The comparison of predictions with field results also included an estimate of uncertainties due to input variables and to measurement error. In addition to the aforementioned comparison, the models' capabilities and limitations were assessed by:

- Carrying out a sensitivity analysis of each model, in order to identify those variables which need to be known with greatest certainty, for reasonable predictions to be obtained.
- Comparing the predictions of the "simple" set of combined models (BED/MPI) with the "complex" set of models (Lectus/Popmite).

Once the models' strengths and weaknesses were identified, the models were then used in a number of scenario modelling, aiming to identify those building design and occupant behaviour features which have the greatest impact on mite

population growth. Furthermore, the models were used in a pilot intervention study (fieldwork: 'Series 3 study') aimed at reducing the HDM allergen exposure for 12 asthmatic children sensitised to dust mite allergens. In this way it was possible to test the use of the models in epidemiological intervention studies.

In summary, the methodology adopted in this thesis aimed at testing the combined hygrothermal population model and assessing its capabilities through a combination of fieldwork and scenarios modelling. The following section describes the fieldwork study in more detail. Some further detailed information on the methods is also provided in the relevant chapters.

4.2 Details of fieldwork

The main objective of the fieldwork was to monitor hygrothermal conditions occurring in real beds, as well as mite survival rates due to those conditions. The results were then compared with the models' predictions. In addition, the Series 3 study included a pilot intervention study on allergen avoidance in asthmatic children. This study also gave the opportunity to test the models deployment in an intervention study.

As illustrated in the previous section, it was considered important to test the validity of the models against data from real beds, occupied by real people. However, monitoring in the field involves working within a range of constraints. For example, beds are a very private location, of which people can be very protective. Part of the methodology involved installing monitoring equipment in beds, or even making some cuts into the mattress, in order to place the equipment. Therefore, recruiting participants and maintaining their co-operation was a hard and time-consuming activity. The monitoring protocol had to take into account the following constraints:

- The interventions and the monitoring activities that the occupants would tolerate (e.g. cutting the mattress).
- Ethical issues: a straightforward way of testing the population models might have been to infest real beds with a known number of mites. However, this is not acceptable ethically.

- Financial issues: although the research project was funded by the EPSRC, some financial constrains still existed for monitoring equipment and for travel.

Following the constraints listed above, the protocol for the fieldwork study had to be developed in order to achieve a balance between the requirements for validating the hygrothermal bed model(s), and the requirements for validating the population model(s). For example, the validation of the mattress models required the monitoring of as many beds and of as many bed locations as possible. On the other hand, in order to guarantee a sufficient time for mite development, the mite bags had to be in place for 6 weeks – ideally during autumn. Since the monitoring equipment was tied up for 6 weeks at a time, the number of monitored beds and bed locations was restricted.

The fieldwork was divided into three successive studies (Series 1, Series 2, Series 3). Series 1 study involved gathering monitoring data (hygrothermal and mite bags) for one bed location (top mattress surface, under the chest area). It also allowed the fieldwork protocol to be tested. Series 2 study involved gathering monitoring data for a larger number of mattress locations (25 locations). Series 3 study was part of an intervention study on allergen avoidance and asthma symptoms in children. Series 3 study also involved gathering monitoring data for 3 locations within mattresses occupied by children. In all studies, the hygrothermal conditions occurring in bedrooms and beds were monitored, together with mite survival rates (mite bags). Twenty five houses were monitored overall in the three fieldwork Series, twenty one of which had mite bags in the participants' beds for a minimum of 6 weeks. Wild mites were used for the mite bags of Series 2 and Series 3. Further details of the 3 fieldwork studies are provided in the following sections.

The fieldwork study was carried out by the author in collaboration with Toby Wilkinson, KTP research associate at Cambridge University and the Medical Entomology Centre. Mr Wilkinson was responsible for the mite bags, whilst the author was responsible for hygrothermal monitoring of bedrooms and beds, as well as for surveying the properties and interviewing the participants. The methodology was approved by University College London's 'Committee on the Ethics of Non-NHS Human Research' (UCL ref.: 0235/001).

The following sections illustrate the 'mite bags' technique, and the three fieldwork studies in more details.

4.2.1 The 'mite bags' technique

The 'mite bags' technique was developed as a way of overcoming sampling and ethical issues related to the monitoring of mites in a real environment. In order to test the population models examined in this thesis, it was necessary to study mite survival rates under varying hygrothermal conditions. In real dwellings mites are exposed to transient conditions, which change on seasonal basis, and daily – particularly as a result of the occupant entering and leaving the bed. However, it is rather difficult to reproduce these conditions in a laboratory setting. In theory, it would be possible to examine mite survival rates under realistic conditions by infesting real beds with a known number of mites and then sampling the live mites after a certain monitoring period. However, this is both unacceptable ethically, and almost impossible to monitor in reality: sampling live mites in real environments can be difficult, since they cling to their substrate and avoid being vacuumed (see Chapter 2). Therefore, the EPSRC-funded team of which the author was part developed the 'mite bags' technique. This involves caging live DP 'wild' mites with food in a mite and allergen proof 'bag' (similar in size and apperance to tea bags, see Fig 4.2.6-7), made from porous material. The 'mite bags' were placed in each monitored bed; after six weeks they were retrieved and the live mites counted. Although the excess food supply and the lack of freedom to move are unrealistic, this technique gives the opportunity to use real occupied beds as 'incubators' (in a way that is acceptable to an Ethical Committee), where mite growth can be examined in relation to real transient conditions, and compared to the population model predictions. In each mite bags location, 3 bags were installed, for repeatability purposes. Each set of mite bags was attached to a hygrothermal sensor, so that it was possible to be reasonably certain about the hygrothermal conditions to which the mites were exposed.

The mite bags were tested for strength, durability and permeability to heat and moisture by Mr Toby Wilkinson (University of Cambridge), who is preparing a paper on these tests. In order to be able to utilise the mite bags in the fieldwork

study, the approval of the UCL Ethics Committee had to be sought, which was obtained successfully.

The mite bags were first tested in the field during the Series 1 study, when 'wild' mites had yet to be reared by the EPSRC-funded team. Therefore, in each of the Series 1 'mite bags', 50 adult laboratory-reared DP mites (1:1 males and females) were encapsulated, together with food (1:1 by weight liver and yeast). In the Series 2 and Series 3 studies, a population of 'wild' DP mites had been gathered. Therefore, in each mite bag utilised in Series 2 and 3, 20 adult *wild* DP mites (1:1 males and females) were encapsulated, together with "wild" food (1:1 by weight skin and dust). Overall, 82 sets of mite bags were installed. However, due to some data loss (e.g. breakage of hygrothermal sensor), 77 sets could be considered for complete data analysis.

4.2.2 'Series 1' Study

Nine dwellings were randomly selected from a database of 1500 dwellings, which had been extensively surveyed as part of the research project: "The Health Impact Assessment of England's New Home Energy Efficiency Scheme" (HEES), rebranded as "WarmFront" (Oreszczyn *et al.*, 2006). The Warmfront study targeted single parents or elderly occupiers. Since the Bartlett (UCL) was a partner in this research project, the author had access to the full data set, including the results of a detailed building and occupant survey, of an air-leakage test (fan-pressurisation) and of previous monitored data. The following describes the data collected in addition to the original Warmfront surveys.

Five of the nine Series 1 dwellings were in the Birmingham area, two in Southampton, one in Liverpool and one in Newcastle. In each of the bedrooms of these dwellings, temperature and relative humidity were monitored for approximately 6 weeks using TinyTag dataloggers (http://www.geminidataloggers.com/)[1]. In each dwelling, a set of mite bags (3 replicate bags in each set) was placed in the participants' beds, on the top mattress surface - corresponding to the area under the sleeper's chest. The surface area

[1] TinyTag Ultra, Part Number 1500. Temperature range: -30 to 50 ^{0}C; RH range: 0 to 95%. Temperature accuracy: +/- 0.2 ^{0}C. RH accuracy: +/- 3%.

under the sleeper's chest - where the mattress equipment was located – was chosen since it has the most varied hygrothermal conditions in the mattress, on a 24-hours cycle. Furthermore, this is the location corresponding to the "bed core", simulated in the BED model (as well as in Lectus).

Hygrothermal loggers were also placed in the bed, near the mite bags (Hobos: www.onset.com)[2]. Since the Hobo dataloggers are similar in size to a box of matches, each logger was modified by putting extension wires to its sensors. In this way the sensors could be placed in the bed under the sleeper's chest without causing any discomfort, while the casing containing the electronic board was located to the side of the mattress. In each mite bag location, two loggers were installed. The sensors of one of the two loggers were encased in a fourth "dummy" mite bag, which contained the same amount of food as the other bags, but no mites. This was done in order to monitor the exact hygrothermal conditions to which the mites were exposed (for example in case the mite food was very hygroscopic). However, when analysing any potential differences between the conditions in a mite bag and those outside a mite bag, these appeared to be smaller than the differences between the two sensors' readings. Nonetheless, having used two sensors rather than one proved useful, since this prevented further data loss when one of the two sensors broke, which unfortunately occurred rather frequently due to the mechanically stressful environment they were located in.

The hygrothermal conditions in the bedrooms and in the beds were logged every 60 minutes. It was decided to carry out the fieldwork study during autumn, since this is the most favourable time for mite growth. This however did not allow enough time to calibrate the Hobo dataloggers. The earliest monitoring started on the 9th Sept. 2004 (Dwelling 1.1) and the latest on the 27th Oct. 2004 (Dwelling 1.9).

The mattress equipment (mite bags and sensors/loggers) was enveloped in a detachable purpose-made pocket made of netting material (Figure 4.2.1-3). This pocket was velcroed to a mite-proof bed cover. During the initial survey visit, the author installed the bed cover with the attached pocket in the participants' bed,

[2] Hobo H8 Series. Temperature range: -20 to 70 ^{0}C; RH range: 25% to 95%. Temperature accuracy: +/- 0.7 ^{0}C at 21 ^{0}C. RH accuracy: +/- 5% over the range of 5 to 50 ^{0}C.

making sure that the mite bags and the sensors were in the area corresponding to the sleeper's chest. The mite-proof bed cover[3] was adopted for three reasons:

1 The mite-proof cover was a safety feature in the unlikely event of a mite bag rupture and therefore was included for ethical reasons.

2 The mite-proof cover offered the equipment (particularly the sensors) some degree of protection from the sleeper's weight and movements.

3 The empirical boundary conditions of the Lectus model had been developed in a lab, where the hygrothermal sensors on the bed surface were protected from direct contact with the sleeper by a mite-proof bed cover. Consequently, the use of a bed cover would help replicate those test conditions, although it was not possible to use the same cover type, since the brand of the cover used during the lab work was not known to the author.

The geographical location of each monitored site meant that it was costly to visit each property several times. Therefore, the detachable pocket containing the mite bags and the sensors was adopted so that participants could easily remove it and send it back via post at the end of the 6 weeks period.

Fig. 4.2.1 Hobo logger with sensor attached to extended wires.

Fig. 4.2.2 Mite-proof cover with detachable pocket containing the mite bags and the hygrothermal loggers.

[3] Jonelle Mattress Protector, 100% Polypropylene. John Lewis, www.johnlewis.com. Part number: 607-30402.

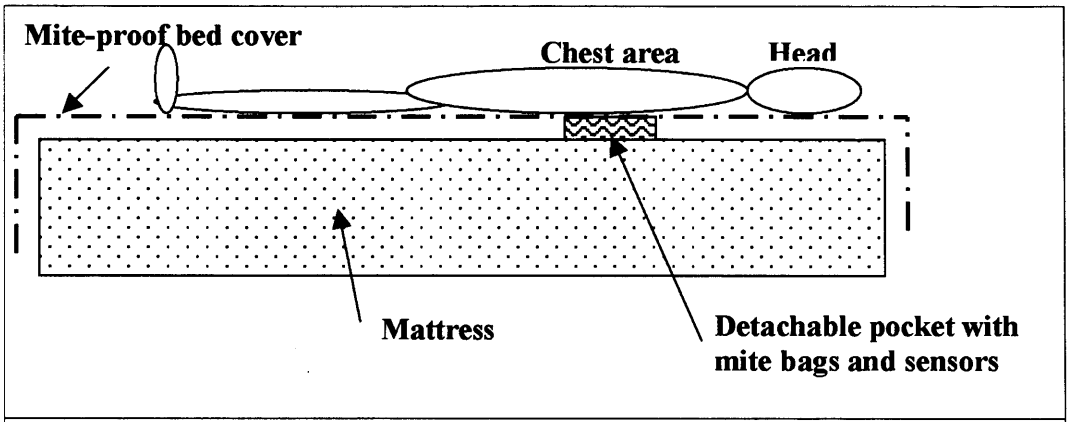


Fig 4.2.3 Schematic cross-section of a Series 1 mattress, with location of mite-proof cover and of detachable pocket.

During the initial survey visit, additional details were gathered on: the mattress (e.g. size, type); the sleeper (e.g. age, gender, weight, typical number of sleep hours per night, smoking and allergy status, etc.); the household heating, ventilation and moisture producing habits. The use of electric blankets or dehumidifiers was also recorded. One of the households (1.9) preferred not to have the mite bags in the bed, and consequently only one hygrothermal logger was placed in Bed 1.9. However, in dwelling 1.1 the bed equipment (mite bags plus sensors) was installed for three different 6 weeks periods. This meant that in Series 1 nine beds and eleven sets of mite bags were monitored overall.

4.2.3 'Series 2' Study

In the Series 2 study, four beds were monitored in greater details, using a 3-dimensional array of 25 hygrothermal sensors, 8 of which were on top of mite bags (3 mite bags in each location). In this way it was possible to build up a more complete picture of how conditions and mite levels may vary within the mattress in real situations. This methodology also allowed for the monitored data to be compared more in details with the Lectus predictions.

The four Series 2 dwellings were selected amongst members of the EPSRC-funded research team, since the methodology adopted in Series 2 was considered too disruptive for members of the general public (e.g. replacing mattress, installing several sensors some of which might cause discomfort). Four identical

sprung mattresses[4] were used and monitored for 6 weeks. The monitoring start times are illustrated in Table 4.2.1

Table 4.2.1 Monitoring times of Series 2 study

Dwelling/Bed Code	Monitoring Start Time
2.1	3 August 2005
2.2	13 September 2005
2.3	6 October 2005
2.4	1 November 2005

Each mattress had 5 layers (see Fig 4.2.5), which in top-to-bottom order were: a first layer of wadding (2.5 cm), a second layer of felt (0.5 cm), a third layer of sprung plus air (14 cm), a fourth layer of felt (0.5 cm), a fifth layer of wadding (2.5 cm). The outer surface of the mattress was finished with a cotton fabric (~0.05cm). In order to make the monitoring results easily comparable with the Lectus predictions, each mattress layer was divided into 5x5 cells. The sensors were then placed in the centre of some cells, in the areas corresponding to: the sleeper's head, chest, feet, sides, and to the mattress edges. In each surface location two sensors were used, since in such locations the sensors could break more easily under the weight and movement of the bed's occupant.

Since the Lectus model was originally developed by monitoring 75 locations in the mattress, ideally the same locations should have been monitored in the Series 2 study. However, the study also aimed to monitor the beds and mite bags in late summer/early autumn, which is the most favourable period for mite growth. This meant that the four Series 2 beds had to be monitored almost at the same time. Since a limited number of dataloggers and sensors was available, the location of the sensors and of the mite bags aimed to balance two aims:

1 Choosing bed locations which would enable a meaningful comparison with the Lectus predictions;

2 Choosing bed locations where a range of hygrothermal conditions occurs, so that the mites in some mite bags thrived, and in other mite bags died.

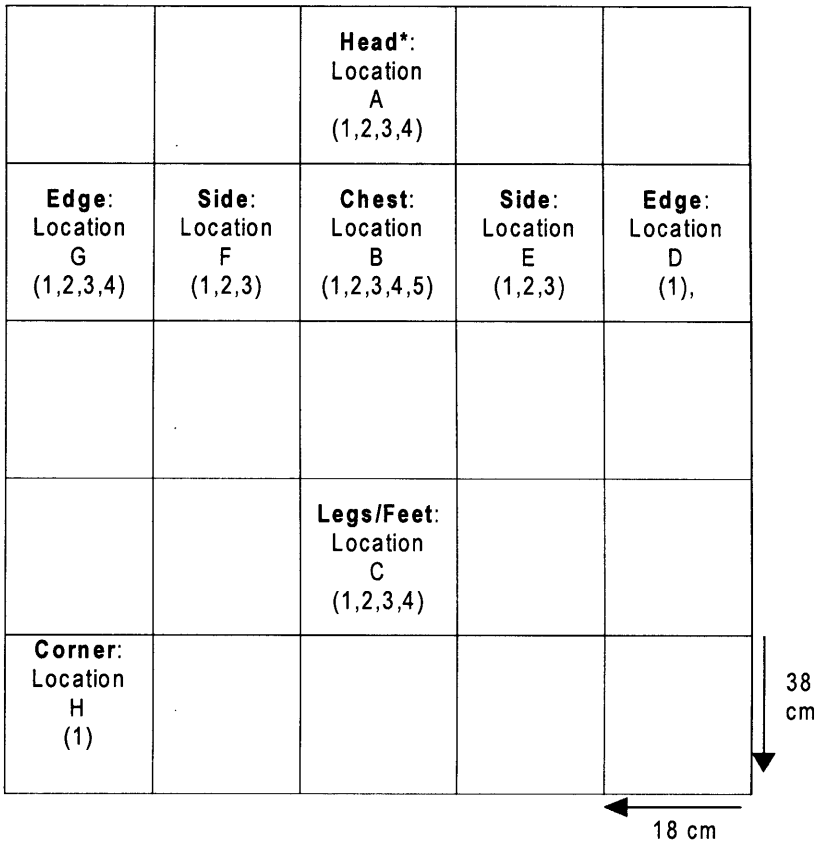
Figure 4.2.5 illustrates the sensors and mite bags positions in a Series 2 mattress. It shows for example that in the area corresponding to the sleeper's chest (location B), 5 sensors were placed: 2 on the top surface (B1/2), 2 in the mattress depths

[4] Simmons, *Orthozone, Premier Backcare*

(B3 and B4), 1 on the bottom surface (B5). In relation to Figure 4.4, the mite bags locations were: A3, B1/2, B3, B4, B5, F3, C1/2, C3. An additional set of three mite bags was also placed in the bedroom, next to the room hygrothermal logger.

Lectus predicts conditions in a single mattress. However, Bed 2.3 was a double bed, occupied by one person, who generally slept on one side of the mattress. Consequently, the preferred side of the bed (correspondent to the width of a typical single mattress) was subdivided in 5x5 cells (Figure 4.2.4). The remaining side of the bed was not monitored. Bed 2.4 was a double bed occupied by two people. In this case the mattress was divided into two equal sides, one of which was subdivided into the 5x5 cells (same as illustrated in Fig 4.2.4); one sensor was placed under the chest of the other bed occupier.

Series 2 Mattress (Top Surface View): Sensors Location



Key of Depth Locations	
1	Top Surface, sensor 1
2	Top Surface, sensor 2
3	between Layer 1 (wadding) and Layer 2 (felt)
4	between Layer 3 (sprung/air) and Layer 4 (felt)
5	Bottom Surface
*	Surface sensors under the pillow

Fig 4.2.4 Schematic representation of sensors location in a Series 2 single mattress

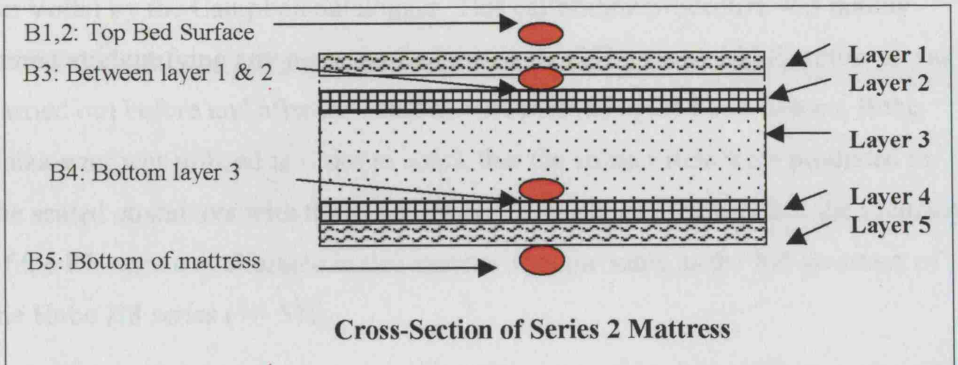


Fig 4.2.5 Schematic cross-section of Series 2 mattress, under the chest area (location B in Figure 4.2.4). The red dots represent the sensors.

The hygrothermal logging was performed using a Campbell Scientific datalogger CR23x (www.campbellsci.co.uk/), which was connected to 2 multiplexers[5] to increase the number of sensors available for logging. In Bed 2.4, the Campbell datalogger CR21x (www.campbellsci.co.uk/) was also adopted.

Two types of sensors were connected to the dataloggers: a thermocouple (Type K thermocouple, RS Components, **rs**www.com)[6] and a RH sensor (Honeywell HIH-3610 Series, RS Components, **rs**www.com/)[7]. The RH sensors were calibrated by placing them first in a sealed container with a saturated NaCl salt solution (75.5% at 25 ^{0}C), and then in a sealed container with a saturated $MgCl_2$ salt solution (32.5% at 25 ^{0}C). A Hobo datalogger was placed in the sealed containers, in order to check that the salt solution was properly mixed, producing the expected RH values[8]. During the calibration period the hobo datalogger registered temperatures between 22-26 ^{0}C in the sealed container. Since the RH produced by saturated salt solutions can vary with changes in temperature, the expected RH produced by the salt solution was adjusted, according to published data on temperature-dependent RH variations for saturated salt solutions (Winston and Bates, 1960). A linear regression function was then calculated for each sensor, by plotting the

[5] AM25T solid state multiplexer, www.campbellsci.co.uk/
[6] Type K thermocouple, RS Components. Temperature range: -40 to 1200 ^{0}C. Temperature accuracy: +/- 1 ^{0}C.
[7] Honeywell HIH-3610 Series, RS Components. RH range: 0% to 90%. RH accuracy: +/- 2% at 25 ^{0}C.
[8] Some salt solutions can be difficult to mix and some initial problems were experienced by the author when mixing the $MgCl_2$ salt solution.

temperature-adjusted RHs given by the salt solutions against the values recorded (in Volts) by the Campbell datalogger. This calibration procedure was mainly aimed at identifying any potential faults with the RH sensors and therefore it was carried out before and after the sensors were utilised in the beds. Since a Hobo datalogger was utilised in order to check that the correct RHs were produced in the sealed containers with the salt solutions, it has been assumed that the accuracy of the RH sensors calibrated in this manner was the same as the RH accuracy of the Hobo H8 series (+/- 5%).

As already mentioned, two sensors were placed in each surface locations, in order to avoid data loss due to sensors' breakages. Despite having calibrated all the RH sensors before and after use, some discrepancies were still found between the readings of those sensors placed in the same bed location. This could be because the calibration was carried out under steady-state conditions, which for example is not representative of differences in response times between sensors. Consequently, averages of the two sensors in one location were taken, in order to calculate the conditions in that location - unless one of the 2 sensors placed in the same location clearly gave unreasonable readings.

The mattress and the sensors for Series 2 were covered with a mite-proof bed cover, identical to the ones used in Series 1. In one of the beds (Bed 2.2), an additional cover had to be used, since the surface sensors proved uncomfortable to that participant. In all beds, the mite bags and the sensors were sewn to the surface of the mattress (for the surface locations), while sticky tape was used to secure the equipment within the mattress depth. Bedroom and outdoor conditions were monitored using Tinytag dataloggers. All hygrothermal data was logged every ten minutes - except in Bed 2.4 when the datalogger CR21x was used, whose smaller memory meant the data had to be logged every 30 minutes. Figure 4.2.6 shows an instrumented Series 2 mattress.

Fig. 4.2.6 Instrumented Series 2 bed.

4.2.4 'Series 3' Study

The Series 3 study was a pilot intervention study on the effectiveness of house dust mite allergen avoidance for twelve asthmatic children (two being controls). In addition to mite allergen removal, the study included tailored advice aimed at reducing mite population growth via changes in moisture production, heating and ventilation habits. Consequently, the study addressed four issues: 1) the effect of allergen removal on the children's health; 2) the effect of tailored advice on

occupant behaviour and the resultant hygrothermal conditions; 3) the effect of the hygrothermal changes on mite populations; and 4) the efficacy of monitoring/modelling techniques. In this thesis the last 3 issues are addressed (Chapter 11), although some aspects of the health impacts are also briefly discussed.

The intervention study obtained additional PPE funds for public dissemination (EP/D064090/1), by means of two 50-minute TV programmes in the 'Dispatches' series, screened on Channel 4 in April 2006 and produced by the project partners 'Twenty Twenty Television'. The role of the research team in the study was mainly to: a) advise the TV producers on scientific matters; b) assess the hygrothermal conditions found in the participants' dwellings and provide tailored advice on how to reduce mite levels via behavioural changes in: moisture production, ventilation and heating habits. Due to its short time-scale and small sample size, the intervention study did not aim to establish the clinical efficacy of allergen avoidance, but rather to illustrate to TV viewers its potential benefits, as well as to give the researchers the opportunity to test the study protocol for a larger future study.

The study also gave an opportunity to install a number of mite bags in the children's beds and bedrooms, thus allowing the comparison between the field results and the population models' predictions. In the Series 3 study, 20 live 'wild' DP mites were caged with food in each mite bag. The mite bags were placed in the child's bedroom (next to the hygrothermal datalogger) and in the child's bed (under the bedding, corresponding to the feet area) - where a sensor was also positioned. The loggers utilised in the beds were the same as those utilised in the Series 1 study (see previous section). However, the sensors and the mite bags were attached directly to the mattress (i.e. no detachable pocket). Three replicate mite bags were placed in each location. In those dwellings which appeared more at risk from mite growth (5 dwellings) – based on the modelling results, see Chapter 11 – additional mite bags and sensors were positioned in the child's bed, under the pillow and under the chest area. The mite bags were placed on the top surface of the mattress, which was then covered with a mite-proof cover[9]. An additional padded cover[10] was also placed on top of the mite-proof

[9] Diagenics micro-porous mite proof bed cover, water vapour permeable (www.diagenics.co.uk).

cover, in order to reduce any discomfort experienced by the children due to the sensors, and in order to prevent sensors' breakages. In addition, it was anticipated that the extra padded cover might protect the mite bags from the body's heat, and therefore give the mites a greater chance of survival. As already mentioned, the hygrothermal conditions recorded in the beds (and in the bedrooms) were utilised in the population models in order to compare the predictions with the mite bags results. The Series 3 hygrothermal data was not utilised to test the hygrothermal bed models, for two main reasons:

1 The study participants for Series 3 were children, with a variety of ages. The BED and the Lectus models had been developed for adults, and it was anticipated that children would have different heat and moisture output during sleep, compared to adults.

2 The additional padded cover (utilised to protect the children from discomfort and the sensors from breakages) represented a confounding factor, when comparing the bed models predictions (for boundary conditions) with the measurement results.

Further detailed information on the study design and protocol is provided in Chapter 11. Figure 4.2.7 shows the equipment utilised in the Series 3 beds.

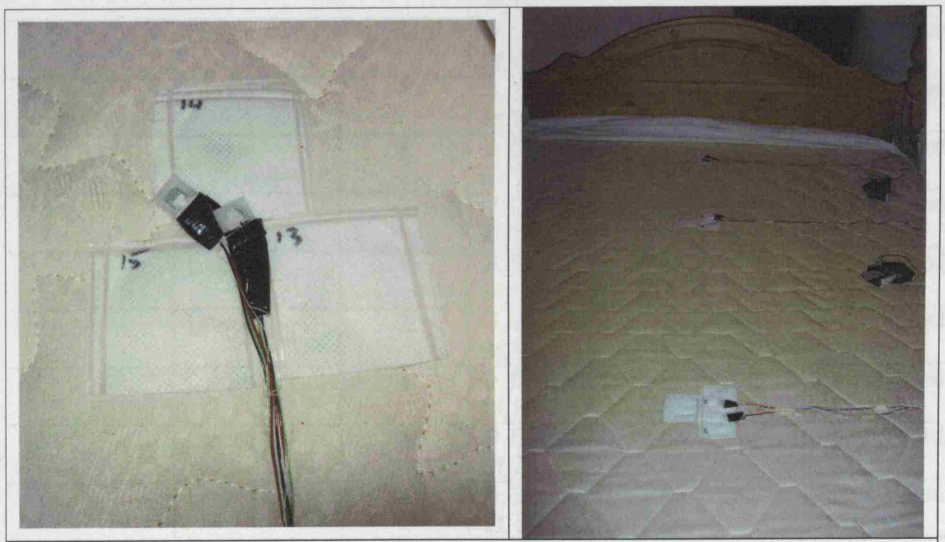


Fig 4.2.7 Mite bags and sensors utilised in the Series 3 study (left) and example of equipment installed in a bed.

[10] John Lewis: Jonelle mattress quilted cover, with 100% cotton cover and 100% polyester filling, code: 607/40202.

Table 4.2.2 is a summary of the fieldwork data.

Table 4.2.2 Summary of Series 1, 2, and 3 fieldwork studies

Dwelling/Bed Code	Dwelling Location	Numb. Occupants (Dwelling)	Hygrothermal Monitoring (O, BR, LR, Bc, Bf, Bh, Bm)*	Mite Bags Location (BR, LR, Bf, Bc, Bh, Bm)*	Monitoring Equipment (H, T, C)#
Series 1					
1.1	Birmingham	1	BR, Bc	Bc	Room: T; Bed: H
1.2	Birmingham	4	BR, Bc	Bc	Room: T; Bed: H
1.3	Newcastle	3	BR, Bc	Bc	Room: T; Bed: H
1.4	Liverpool	2	BR, Bc	Bc	Room: T; Bed: H
1.5	Southampton	1	BR, Bc	Bc	Room: T; Bed: H
1.6	Southampton	5	BR, Bc	Bc	Room: T; Bed: H
1.7	Birmingham	2	BR, Bc	Bc	Room: T; Bed: H
1.8	Birmingham	1	BR, Bc	Bc	Room: T; Bed: H
1.9	Birmingham	1	BR, Bc	(none)	Room: T; Bed: H
Series 2					
2.1	Cambridge	2	O, BR, LR, Bm	BR, Bm	Room: T; Bed: C
2.2	Milton Keynes	3	O, BR, LR, Bm	BR, Bm	Room: T; Bed: C
2.3	Cambridge	3	O, BR, LR, Bm	BR, Bm	Room: T; Bed: C
2.4	London	2	O, BR, LR, Bm	BR, Bm	Room: T; Bed: C
Series 3					
1	London	4	O, BR, LR, Bc, Bf, Bh	BR, Bf, Bc, Bh	Room: T; Bed: H
2	London	2	O, BR, LR, Bc, Bf, Bh	BR, Bf,	Room: T; Bed: H
3	London	2	O, BR, LR, Bc, Bf, Bh	BR, Bf,	Room: T; Bed: H
4~	London	3	O, BR, LR, Bc, Bf, Bh	BR, Bf,	Room: T; Bed: H
5	London	4	O, BR, LR, Bc, Bf, Bh	BR, Bf,	Room: T; Bed: H
6	London	5	O, BR, LR, Bc, Bf, Bh	LR, BR	Room: T; Bed: H
7	London	8	O, BR, LR, Bc, Bf, Bh	BR, Bf, Bc, Bh	Room: T; Bed: H
8	London	3	O, BR, LR, Bc, Bf, Bh	LR, BR	Room: T; Bed: H
9	London	4	O, BR, LR, Bc, Bf, Bh	BR, Bf,	Room: T; Bed: H
10	London	2	O, BR, LR, Bc, Bf, Bh	BR, Bf, Bc, Bh	Room: T; Bed: H
11	London	4	O, BR, LR, Bc, Bf, Bh	BR, Bf, Bc, Bh	Room: T; Bed: H
12a	London	4	O, BR, LR, Bc, Bf, Bh	BR, Bf, Bc, Bh	Room: T; Bed: H
12b	London	4	O, BR, LR, Bc, Bf, Bh	BR, Bf, Bc, Bh	Room: T; Bed: H

* **O**: Outdoor; **BR**: Bedroom; **LR**: Living Room; **Bc**: Bed's top surface, chest area; **Bf**: Bed's top surface, feet area; **Bh**: Bed's top surface, under pillow area; **Bm**: Bed, multiple locations (see Fig 4.2.4).

H: Hobo H8 Series; **T**: TinyTag Ultra; C: Campbell Datalogger (23x or 21x), plus Type K thermocouple and Honeywell HIH-3610 Series (RH sensor).

~ Post-intervention data could not be retrieved in this house.

Chapter 4: References

Oreszczyn T, Ridley I, Sung H, Wilkinson P and the Warm Front Study Group, (2006), Mould and winter indoor relative humidity in low income households in England, *Indoor and Built Environment*, 15: 125-135.

Winston P, Bates D, (1960), Saturated solutions for the control of humidity in biological research, *Ecology*, 41(1): 232-237.

CHAPTER 5:

COMPARISON OF FIELDWORK RESULTS WITH LECTUS PREDICTIONS

CHAPTER 5: COMPARISON OF FIELDWORK RESULTS WITH LECTUS PREDICTIONS

5.1 Introduction

This chapter discusses the comparison of the Lectus (Ridley *et al.*, submitted) predictions with the results of the fieldwork study (Series 1 and 2). The Lectus model - already illustrated in Chapter 3 – is based on 3 main elements, which are addressed separately in each section:

1. The empirical boundary conditions, which were originally based on the results of 10 volunteers each sleeping for one night in a bed located in a laboratory (section 5.2).
2. The rate at which bed conditions change, when the sleeper gets into bed, and when the sleeper leaves the bed. This was also derived from the lab tests (section 5.3).
3. The heat and moisture transfer calculations for each cell within the depth of the mattress, which is also affected by the hygrothermal properties of the mattress materials (section 5.4).

The chapter ends with a summary discussion (section 5.5).

5.2 Boundary conditions in Lectus and measured bed surface data

In Lectus the conditions on the surface of the mattress are predicted by using both room conditions and an empirical model based on the results of an experimental test bed. During the original experiments for the Lectus model[1], a foam and a sprung mattress were investigated, with 10 volunteers each sleeping in both beds, for approximately 8 hours at a time. The surface of the Lectus mattress was divided into 25 zones (Figure 5.2.1). At all times other than when the bed was occupied and on all surfaces other than the top, the boundary conditions were assumed to be the same as the room. When the bed is occupied, the top surface

[1] First EPSRC-funded research project, 2000-2002, in which the author of this thesis was not involved.

conditions in each zone were assumed to be those illustrated in Figure 5.2.1. For example, it was assumed that on the surface zone corresponding to the chest area, the temperature is 34 °C and the Vapour Pressure Excess (VPX) is 1000 Pa (when the bed is occupied). The surface vapour pressure in each zone is then calculated by adding its VPX to the room's vapour pressure. These assumptions were developed by calculating the average conditions measured on the top surface of the laboratory mattress, when the bed was occupied by 10 volunteers (one at a time). It should be highlighted that the conditions under the head area refer to conditions *under* the pillow.

In the following sections, the boundary conditions assumed in Lectus (for a sprung mattress) will be compared with the conditions measured in the fieldwork studies (Series 1 and 2). In particular, the results measured under the chest area (from the Series 1 and Series 2 studies) will be analysed in section 5.2.1, followed by the results from the 'still test' in Series 2 (section 5.2.2.). Section 5.2.3 includes a comparison of the results from all the data-sets, and section 5.2.4 is a summary on the boundary conditions analysis.

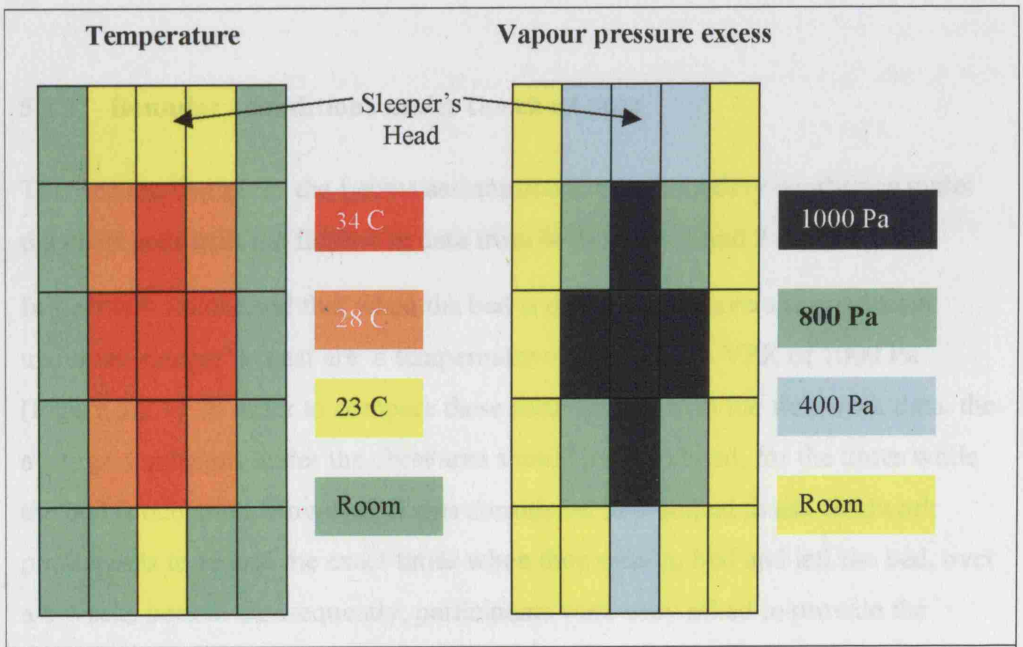


Fig. 5.2.1 Top of the mattress boundary conditions in Lectus, for an occupied sprung mattress.

Chapter 4 described in details the protocol followed for the fieldwork study. For the purposes of this section, it may be useful to highlight some elements of the

fieldwork protocol. Firstly, in the Series 1 study, sensors were placed on the mattress surface - together with 3 mite bags - under the sleeper's chest, for 6 weeks, in 8 beds[2] (hourly values). Bedroom conditions were also monitored. Participants from Series 1 were randomly selected from the Warmfront study (Oreszczyn *et al.*, 2006), which targeted single parents or elderly people, and aimed to assess the health effect of energy efficiency measures. The mattresses in Series 1 were all sprung, from different brands. The duvet and the pillow were also from different brands.

In Series 2, four additional beds were monitored in more detail than in Series 2, using a 3-dimensional array of 25 hygrothermal sensors, 8 of which were placed on the bed surface, as illustrated in Figure 4.2.4-5. Conditions in the bed and bedroom were logged every 10 minutes. In Series 2 the same brand and model of mattress was used. Duvet and pillows were not standardised. Participants for Series 2 were team members of the research team or their families (age range: 14-60 years old). As already mentioned, Chapter 4 provides further details on the protocol for Series 1 and Series 2.

5.2.1 Boundary conditions under the chest area

This section compares the Lectus assumption for the boundary conditions under the chest area with the fieldwork data from both Series 1 and 2 studies.

In Lectus it is assumed that when the bed is occupied, the average conditions under the sleeper's chest are: a temperature of 34 °C and a VPX of 1000 Pa (Figure 5.2.1). In order to compare these assumptions with the fieldwork data, the average conditions under the chest area should be calculated, for the times while the bed is occupied. However, it was considered impractical to ask fieldwork participants to record the exact times when they went to bed and left the bed, over a 6 weeks period. Consequently, participants were only asked to provide the average number of hours of sleep per night. It was thought that it would be quite clear when the bed was occupied, by analysing the plots from the monitored bed conditions.

[2] Nine beds were monitored, but the bed sensors in one of the 9 beds produced unreliable data, which had to be disregarded.

Figure 5.2.2 shows the conditions corresponding to three days of monitoring for a Series 1 bed (Bed 1.4), where it is rather clear when the bed is occupied. The graph also shows that during occupation the temperature only reaches 31-32 ºC. This could be due, for example, to the participant sleeping slightly to the side of the sensor, and/or to the participant wearing insulating sleeping clothes.

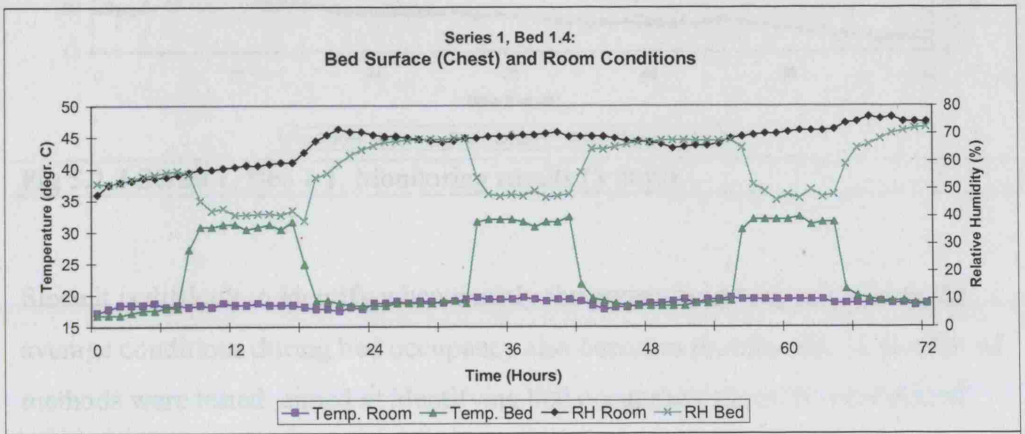


Fig. 5.2.2 Series 1, Bed 1.4. Monitoring results (3 days).

On the other hand, by analysing the plots from other beds, in some cases it was less clear when the bed was occupied. For example, in Figure 5.2.3 the results for Bed 1.1 show rather variable conditions. These are probably a combination of the participant moving in the bed, and leaving/coming back to the bed rather frequently. The variable conditions illustrated in Figure 5.2.3 also show that bed conditions under the chest are likely to be variable *within* individuals, as well as *across* individuals – as suggested by published information (see Chapter 2). The conditions illustrated in Figure 5.2.3 are somewhat "extreme", since they correspond to a participant who spent long hours in bed for health reasons[3]. However, they illustrate how hygrothermal conditions on the top surface of an occupied mattress can be at times variable.

[3] Participant 1.1 was the only one who spent unusually long hours in bed. Table 5.2.1 illustrates the average number of hours in bed per day.

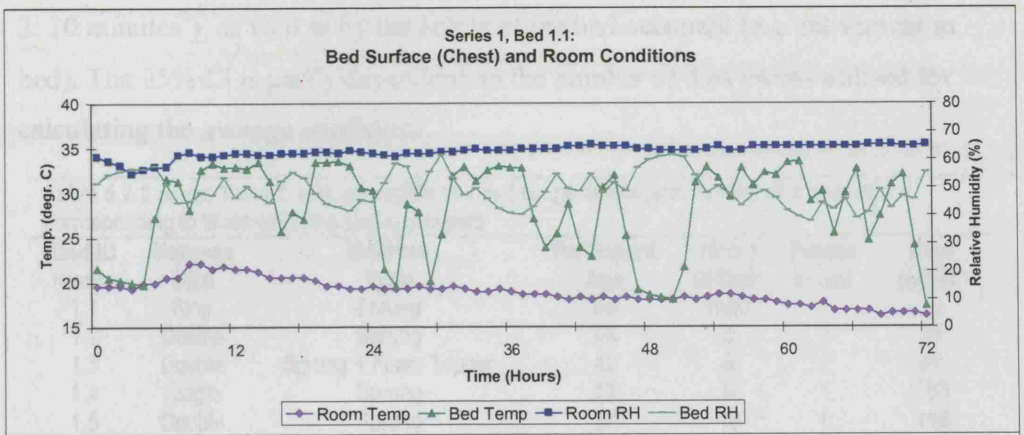


Fig 5.2.3 Series 1, Bed 1.1. Monitoring results (3 days).

Since it is difficult to identify when exactly the person is in bed, calculating the average conditions during bed occupancy also becomes problematic. A number of methods were tested, aimed at identifying bed occupancy times. It was decided that the most effective (and less time-consuming) method for identifying data from times when the bed is occupied was selecting data from 3:00 am to 5:00 am, when most people are asleep. In order to avoid the inclusion of data corresponding to times when the participant might have briefly left the bed during that time (or for example left the bed earlier than 5:00 am), data between 3:00 am and 5:00 am was excluded, if the monitored bed temperature was below 28 °C. This temperature threshold value for bed occupation was identified by analysing the plots from several monitored beds. Of course, a higher threshold value might have been selected, however this may have excluded data when the participant is in bed, but he/she is not on top of the sensor.

Once the data corresponding to occupied bed times was identified, the average temperature and VPX under the chest area were calculated for each Series 1 and 2 bed. The standard deviation and the 95% Confidence Intervals (CI) were also calculated. In this way, the variability in hygrothermal conditions under the chest was identified for each participant. Table 5.2.1 gives details of the mattress type, size and occupancy for the twelve Series 1 and Series 2 beds. It also provides the total number of data points which were calculated as "bed occupied times" (i.e. between 3:00 am and 5:00 am, temperature above 28 °C). The number of selected data points depends on the logging frequency of the data (Series 1: 1 hour; Series

2: 10 minutes[4]), as well as by the habits of the bed occupant (e.g. movement in bed). The 95% CI is partly dependent on the number of data points utilised for calculating the average conditions.

Table 5.2.1 Series 1 and 2: bed, participant and bed usage details, and number of data points corresponding to times when the bed is occupied

Bed ID Numb.	Mattress Size	Mattress Type	Participant Age	Hours in bed~	People in bed	Data points*
1.1	King	Sprung	68	5-20	1	26
1.2	Double	Sprung	58	5	2	33
1.3	Double	Sprung + Foam Topper	42	8	1	87
1.4	Single	Sprung	53	8	1	156
1.5	Double	Sprung	67	10	1	118
1.6	Double	Sprung	34	8	2	118
1.7	Double	Sprung	77	10	1	72
1.8	Double	Sprung + Feathers Topper	80	8	1	116
2.1	Single	Sprung, *Premier BackCare*	60	7	1	533
2.2	Single	Sprung, *Premier BackCare*#	14	7	1	468
2.3	Double	Sprung, *Premier BackCare*	30	8	1	483
2.4	Double	Sprung, *Premier BackCare*	32	8	2	78

* Number of data points corresponding to the times when the bed is occupied (i.e. between 3:00 am and 5:00 am, temperature above 28 °C); ~Average number of hours in bed per day, from interview; # In bed 2.2 an additional padded cover was placed on top of the sensors, since the latter caused discomfort to the participant.

Figure 5.2.4 shows a plot of the average temperature (and correspondent 95% CI) monitored during occupancy times under the chest, for the twelve Series 1 and Series 2 beds. The Lectus assumption of 34 °C is also plotted in the graph, as well as the total average temperature (and 95% CI) calculated from the averages of the 12 beds.

[4] However in bed 2.4 conditions were initially monitored every 30 minutes, as a datalogger with a smaller memory was used (Campbell, CR21x). Half way through the monitoring the datalogger was replaced with one with a larger memory (Campbell, CR23x) so that conditions could be monitored every 10 minutes.

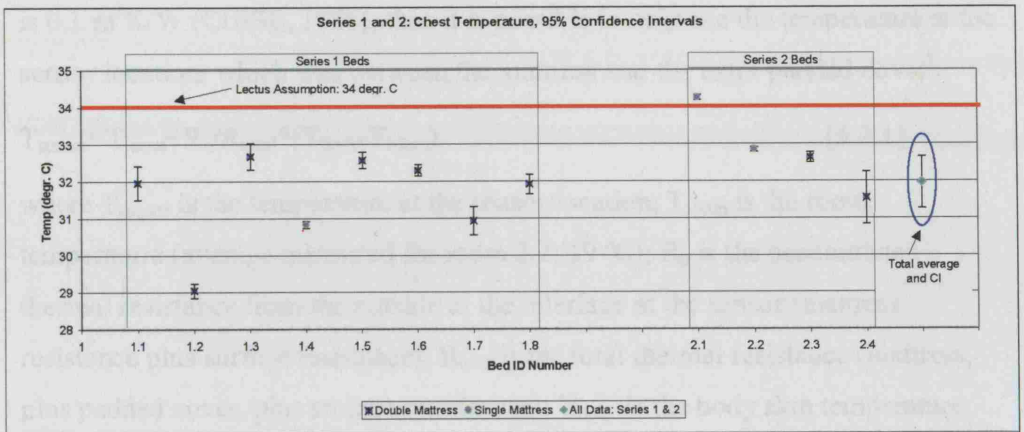


Fig 5.2.4 Average and 95% CI temperature under the chest (occupied bed), for Series 1 and Series 2 beds.

Figure 5.2.4 shows that for the majority of the field beds, the 95% CIs for the temperature under the chest are below the Lectus assumption of 34 °C. The total 95% CI calculated from all the twelve beds is also below the Lectus assumption. In Figure 5.2.4 double and single mattresses are also identified and it appears that the 95% CI is smaller for the 3 single beds. This is most probably because there is less scope for movement in a single bed, which results in a smaller variability in the temperature, when the bed is occupied. Nonetheless, only one of the 3 single mattresses (and of the 12 beds) has an average temperature slightly above the Lectus assumption.

It should also be mentioned however that in one of the 3 single mattresses (bed 2.2), an additional padded cover had to be placed on top of the sensors, since the latter caused discomfort to the participant. This represents a confounding factor when assessing the boundary conditions from bed 2.2 and it is particularly unfortunate, since the same mattress brand and model was used for bed 2.1 and 2.2, while bed 1.4 used a different mattress brand. However, the effect of the extra padded cover in bed 2.2 on the bed temperatures measured while the bed was occupied can be estimated under steady-state conditions. The extra padded cover in bed 2.2 had a thickness of approximately 1 cm and was made of wool, whose thermal conductivity can be assumed as 0.04 W/mK (www.engineeringtoolbox.com). If it is assumed that: the surface temperature of the skin is 34 °C (Fanger, 1970); the mattress thermal conductivity is 0.06 (as assumed in the BED model) with a thickness of 0.2 m; and the surface resistance

is 0.1 m^2K/W (CIBSE, 1986), then it is possible to estimate the temperature at the sensor location, which was between the mattress and the extra padded cover[5].

$$T_{sensor}=T_{room}+R_n/R_{total}*(T_{body}-T_{room}) \quad [5.2.1]$$

where T_{sensor} is the temperature at the sensor location; T_{room} is the room temperature (average measured for room 2.2: 19 °C); R_n is the accumulated thermal resistance from the outside to the interface at the sensor (mattress resistance plus surface resistance); R_{total} is the total thermal resistance (mattress, plus padded cover, plus surface resistances); T_{body} is the body skin temperature, assumed as 34 °C.

By using the assumptions and formula above, the temperature at the sensor location is estimated as 32.98 °C. This figure is very close to the average temperature measured while the bed 2.2 was occupied: 32.85 °C. This also suggests that in bed 2.2 the effect of movement might have been small on average - which is not surprising, since bed 2.2 was a single bed. A similar calculation to estimate the effect of the extra padded cover on the vapour pressure at the sensor location is more difficult, since moisture production from the body is not always constant, but it can vary for example for thermoregulatory purposes.

Figure 5.2.5 shows a plot of the average VPX (and correspondent 95% CI) monitored during occupancy times under the chest, for the twelve Series 1 and Series 2 beds. The Lectus assumption of 1000 Pa is also plotted in the graph, as well as the total average VPX (and 95% CI) calculated from the averages of the 12 beds.

[5] A mite-proof cover was used in all beds between the body and the sensor. However, this cover is ignored in these calculations, since its thermal resistance is assumed negligible.

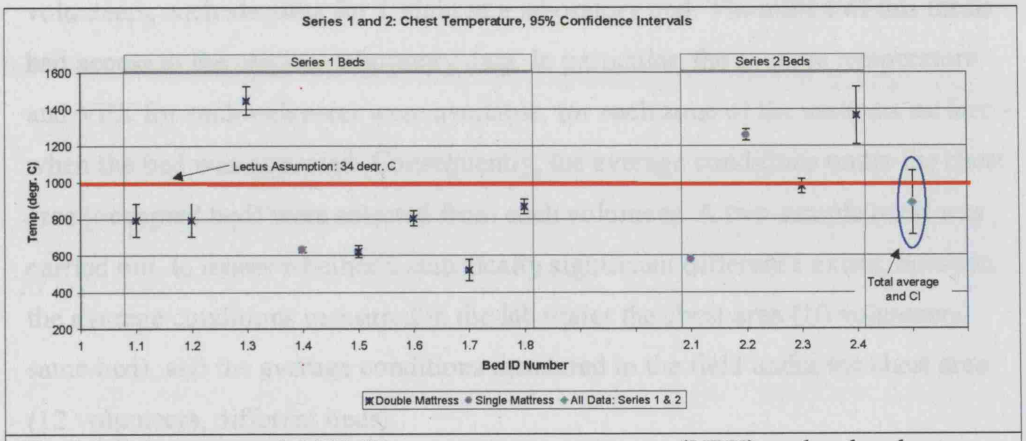


Fig 5.2.5 Average and 95% CI vapour pressure excess (VPX) under the chest (occupied bed), for Series 1 and Series 2 beds.

Figure 5.2.5 shows that three out of the twelve participants have a VPX above the Lectus assumption, one participant has a 95% CI which include the Lectus assumption and the remaining participants are below such assumption. The total 95% CI calculated from all the twelve beds includes the Lectus assumption, although by a small amount. Again, the three single mattresses appear to have smaller 95% CIs.

Figure 5.2.4 and 5.2.5 show that there are some discrepancies between the Lectus assumptions for conditions under the chest, and the fieldwork data – particularly the temperature. However, the Lectus assumptions were developed for single mattresses, while the majority of the fieldwork beds are double mattresses with 1 occupant, which result in a greater scope for movement in the bed. This confounding factor makes it difficult to assess whether the discrepancy between the field data and the Lectus assumptions may be due, for example, to: very large variability in hygrothermal conditions between participants, and/or to the effect of different mattresses types (Series 1 data), and/or the effect of greater scope for movement in double beds, and/or the effect of different types of duvets on the boundary conditions. The question arises as to whether comparing data from the Series 1 and 2 studies with the Lectus assumption is appropriate. In order to assess this, it was decided to directly compare the fieldwork data with the data from the original laboratory work, which was utilised to develop the Lectus assumptions.

As already mentioned, the Lectus assumptions were developed by monitoring 10 volunteers, each sleeping for 1 night in a laboratory bed. The author of this thesis had access to the original laboratory data. In particular, the average temperature and VPX for each volunteer were available, for each zone of the mattress surface - when the bed was occupied. Consequently, the average conditions under the chest area (occupied bed) were selected from each volunteer. A two-sample t-test was carried out, to assess whether a statistically significant difference exists, between the average conditions measured in the lab under the chest area (10 volunteers, same bed), and the average conditions measured in the field under the chest area (12 volunteers, different beds).

Table 5.2.2 illustrates the results of the t-test for the temperature data under the chest. The results suggest that the difference between the temperature means from the two samples (lab and field) is statistically different, and should therefore be considered as representative of 2 different populations. However, if the t-test is performed by excluding the double-beds results from the field data, the 95% CI becomes wider and the p-value is 0.4 (Table 5.2.3), indicating that the difference between the temperature means of the two samples is not statistically significant. This result is however influenced by the small sample size of the 3 single mattresses in the field sample.

It should also be pointed out at this stage that the 95% CI for the lab data is 32.7-34.4 °C, with an average of 33.6 °C, and the Lectus assumption for the temperature under the chest rounds this figure to 34 °C.

Table 5.2.2 Laboratory temperature data compared with field temperature data: two-sample t-test

	Sample size	Mean (°C)	St. Dev. (°C)	95% CI (°C)	p-value*
Lab	10	33.6	1.5	32.7-34.4	0.02
Field	12	32.0	1.3	31.2-32.7	

*The p-value was calculated for a t-test assuming equal variance, and for a t-test assuming unequal variance. No significant difference was found between the 2 assumptions.

Table 5.2.3 Laboratory temperature data compared with field temperature data (3 single mattress only): two-sample t-test

	Sample size	Mean (°C)	St. Dev. (°C)	95% CI (°C)	p-value*
Lab	10	33.6	1.5	32.7-34.4	0.4
Field	3	32.7	1.7	30.7-34.6	

*The p-value was calculated for a t-test assuming equal variance, and for a t-test assuming unequal variance. No significant difference was found between the 2 assumptions.

Table 5.2.4 illustrates the results of the t-test for the vapour pressure excess data under the chest. The p-value is 0.1, indicating that the difference between the VPX means of the two samples is not statistically significant.

Table 5.2.4 Laboratory temperature data compared with field vapour pressure excess data: two-sample t-test

	Sample size	Mean (Pa)	St. Dev. (Pa)	95% CI (Pa)	p-value*
Lab	10	1164.0	467.1	899.8-1428.3	0.1
Field	12	888.1	312.8	711.2-1065.1	

*The p-value was calculated for a t-test assuming equal variance, and for a t-test assuming unequal variance. No significant difference was found between the 2 assumptions.

The results from the t-tests comparing the lab-data with the field data suggest that the presence of double beds (i.e. more scope for movement in bed) in the field-sample may represent a significant confounding variable when comparing the Lectus temperature assumption for the chest area with the field data. This is true for the temperature data, while the VPX results are not affected – most probably because the mattress properties are such that movement in bed causes more frequent/larger variations in temperature, than variations in VPX.

This section aimed to compare the Lectus assumption for the boundary conditions under the chest area with the fieldwork data from Series 1 and from Series 2. The data analysis revealed that the field data is significantly different from the Lectus assumption, for the temperature results. This is most probably due to the majority of the field beds being double mattresses, which gives the occupant a larger scope for movement in bed – whilst the Lectus assumptions were developed for single beds. The next section examines the results from a test where participants from Series 2 beds were asked to stay in bed totally still for at least 20 minutes ('still test'). The results from the 'still test' are then compared with the results from the original lab-work, from which the Lectus assumptions were developed.

5.2.2 Results from the 'Still Test' in the Series 2 study

In the previous section it emerged that one of the main confounding factors in the analysis of the field data was the sleeper being able to move quite a lot whilst occupying a double bed. Consequently, it was decided to perform a 'still test' in the Series 2 study, where the participants were asked to stay totally still in the

monitored bed for at least 20 minutes. The exact times when the bed was occupied during the 'still test' were recorded, and the hygrothermal conditions of bed and bedroom were logged every minute – as opposed to the 10 minutes of the "standard" Series 2 study. Participants were asked to wear similar clothing for the 'still test' to those used whilst sleeping. They were also instructed to leave the duvet on or off as they preferred, according to whether they felt warm or cold during the test. This replicates what happens in reality during sleep, and it also reproduces the conditions of the original lab experiments for the Lectus boundary conditions (where volunteers could remove the duvet at their will).

The Lectus boundary conditions represent the average temperature and VPX occurring in each surface zone of the mattress, when the lab-bed was occupied by 10 volunteers, one at a time. Consequently, the average conditions occurring during the 'still test' in each monitored surface location of the Series 2 beds were calculated, and compared with the Lectus assumptions.

Figure 5.2.6 shows the hygrothermal profile of Bed 2.1 (under chest) during the 'still test', and it appears that at least 10-15 minutes are required for the hygrothermal conditions to become more stable. Therefore, the averages of all the monitored data from the 'still test' may not be the appropriate parameters to be compared with the Lectus boundary conditions, and consequently the values recorded *at the end* of the bed occupancy period could be considered as more representative of steady-state values. This however assumes that 20 minutes was a sufficient time to reach state-state conditions. Unfortunately, it was considered unrealistic to ask the participants to perform the still test for longer periods. However, Figure 5.2.6 shows that the greatest variations in hygrothermal conditions occurred within the first 15 minutes.

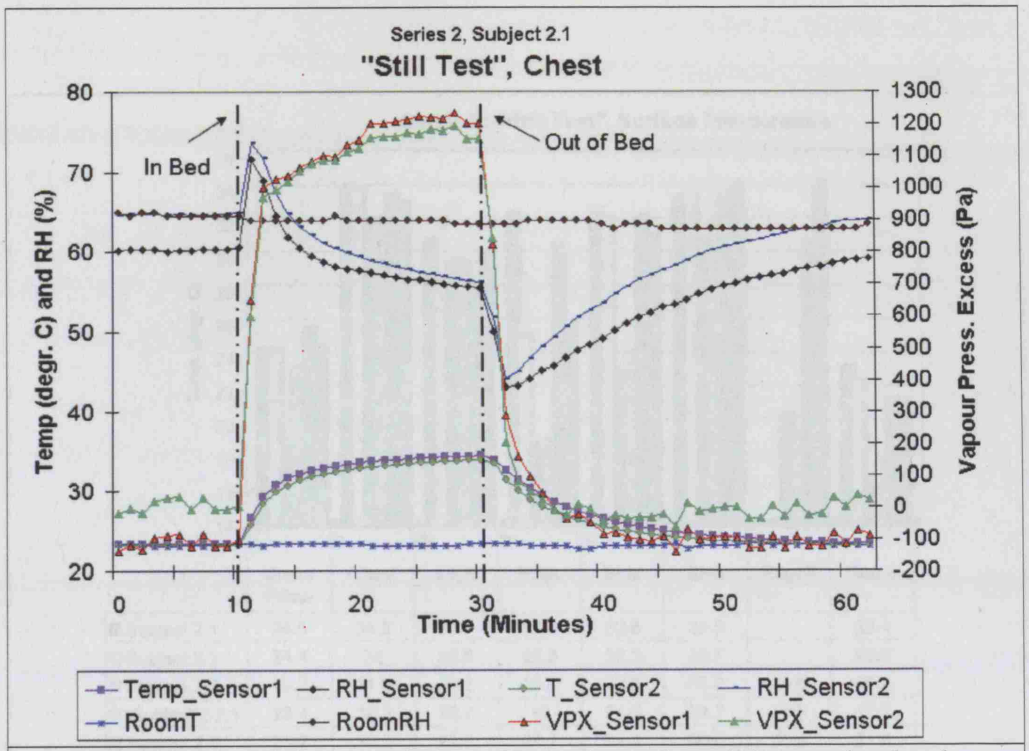
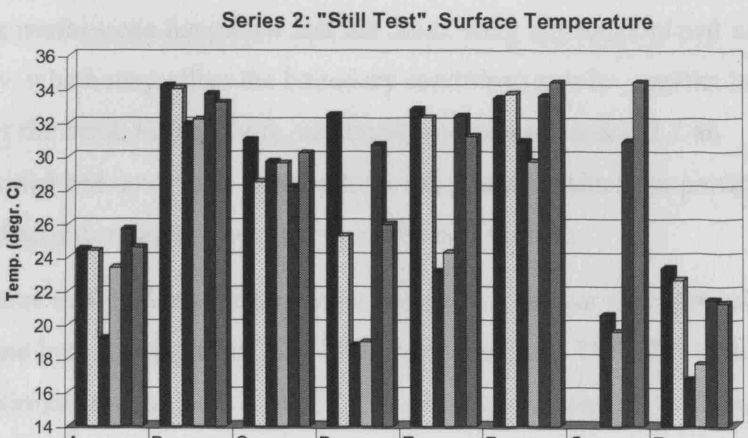
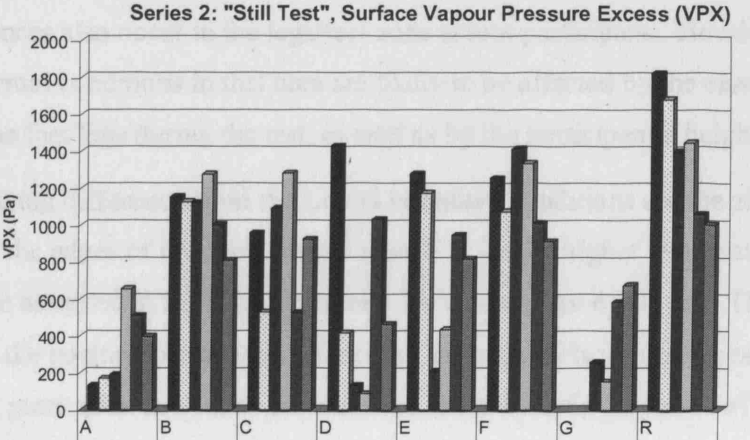


Fig 5.2.6 Series 2, 'Still Test', Bed 2.1: Hygrothermal conditions under the chest.

It should be mentioned at this stage that no 'still test' could be carried out for Bed 2.3. However, in each of the other three 'Series 2 beds', an additional participant - other than the one monitored for 6 weeks – performed the 'still test', in order to assess any potential differences in heat and moisture outputs during bed occupancy. The main Series 2 participants are here referred to as: 2.1, 2.2, 2.4, while the correspondent extra subjects are respectively indicated as: 2.1.1; 2.2.1; 2.4.1. Figure 5.2.7 shows the results of the "last reading" in the 'still test', before the bed was vacated, for the six participants of the 'still test' (three Series 2 beds).



	A Under Pillow	B Chest	C Legs	D Edge	E Side	F Side	G Edge#	R Room
Subject 2.1	24.5	34.2	31	32.5	32.8	33.5		23.4
Subject 2.1.1	24.4	34	28.5	25.3	32.3	33.7		22.7
Subject 2.2	19.2	31.9	29.7	18.8	23.2	30.9	20.6	16.8
Subject 2.2.1	23.4	32.2	29.6	19	24.3	29.7	19.6	17.7
Subject 2.4	25.7	33.7	28.2	30.7	32.4	33.6	30.9	21.5
Subject 2.4.1	24.6	33.2	30.2	26	31.2	34.4	34.4	21.3



	A Under Pillow	B Chest	C Legs	D Edge	E Side	F Side	G Edge#	R Room*
Subject 2.1	132.1	1151.4	956.1	1428.2	1276.7	1253.8		1823.7
Subject 2.1.1	168.6	1124.2	529	414.6	1173	1073.4		1682
Subject 2.2	189.1	1118.5	1090.4	130.6	207.6	1412.3	256	1398.5
Subject 2.2.1	652.3	1274.4	1280.8	86.5	433.5	1333.5	150.9	1445.3
Subject 2.4	506.7	1001	523.9	1030.9	945.1	1009.3	583.7	1059.1
Subject 2.4.1	396.5	808.8	922.2	462.6	815.2	911.8	672.9	1002

Fig 5.2.7 Series 2, 'Still Test'. Bed surface hygrothermal conditions at the end of the bed occupancy period, for each monitored zone (illustrated in Fig. 4.2.2). Note: #G was not monitored in bed 2.1; * All vapour pressures are excess, (VPX), except for room values (Pa).

Figure 5.2.7 gives the opportunity to compare 2 subjects who performed the 'still test' in the same bed, one at a time (2.1 versus 2.1.1; 2.2 versus 2.2.1; 2.4 versus 2.4.1). This is useful since the pillow and the duvet were not standardised in Series 2 study, which may affect the boundary conditions results – particularly for the area under the head. Furthermore, as already mentioned in Bed 2.2 an additional padded bed cover was used on top of the sensors, since the participant experienced discomfort due to the sensors on the bed surface.

The results show that the temperature under the chest is similar amongst subjects within the same bed, varying from 32-34 °C across the beds. The VPX under the chest is very similar for subjects 2.1 and 2.1.1 – which were tested on the same bed – and for subject 2.2.

The temperature under the pillow is fairly similar for all subjects, except for subject 2.2. However, the VPX under the pillow is fairly dissimilar across the beds, with some differences between subjects in the same bed. Differences in pillow types (i.e. hygrothermal properties) may partly account for these variations.

Some differences also occur in the legs/feet zone across participants. However, the hygrothermal conditions in that area are likely to be affected by the exact position of the feet/legs during the test, as well as by the participant's height.

The most striking difference from the Lectus boundary conditions are the zones to the sides and the edges of the chest, which appear to have a higher temperature and VPX than assumed in Lectus, particularly for some of the 6 subjects. This suggests that the position of the arms affects the mattress surface conditions. Furthermore, participant 2.4.1 may have had his chest towards the centre of the double bed – rather than in the centre of one half of the bed – since the temperature on the edge and side is higher than the one recorded for the chest sensor.

Since the exact position of the arms, the feet, etc. appear to significantly affect the hygrothermal conditions of some surface zones, this suggests that the monitored hygrothermal conditions can be rather different if a participant's body part is a little off a sensor (or is exactly on a sensor). If that is the case, the values given by a sensor should be taken with some caution, when extending them to the whole zone "represented" by the sensor.

Table 5.2.5 shows the average and 95% CI for the temperature and VPX calculated in each bed surface zone from the 6 participants for the 'still test'. The Lectus assumptions for each zone are also indicated.

Table 5.2.5 Average (and 95% CI) conditions calculated for the 6 participants to the 'still test' in Series 2, in each monitored bed zone. Comparison with the Lectus assumptions.

Zones monitored in Series 2 *(ref. Fig 4.2.2.)*	Temp. (°C), Average	Temp. (°C), 95% CI	Lectus Assumption, (°C)	VPX (Pa), Average	VPX (Pa), 95% CI	Lectus Assumption, (Pa)
Bed: A (Under Pillow)	23.6	21.8-25.4	23	340.9	171.6-510.2	400
Bed: B (Chest)	33.2	32.4-34.0	34	1079.7	952.6-1206.8	1000
Bed: C (Legs/Feet)	29.5	28.7-30.3	34	883.7	640.4-1127.0	1000
Bed: E,F* (Side)	31.0	28.5-33.5	28	987.1	741.1-1233.1	800
Bed: D,G~ (Edge)	(6.0)#	(2.5-9.4)#	(0)#	504.1	191.0-817.1	0

Note: In this table the results of the still test correspond to the average of the "last reading" monitored for each of the 6 participants, before leaving the bed. #Temperature difference between bed and room. * Average of zone E and F. ~Average of zone D and G.

The results in Table 5.2.5 indicate that the conditions under the pillow monitored during the 'still test' compare favourably with the Lectus assumptions, since the 95% CI include such assumptions. This is also true for the chest area, although the Lectus assumption for the temperature may be a little too high. On the other hand, the Lectus assumption for the temperature appears a little low for the side areas. For the edge areas, the Lectus assumptions may also be a little low for both temperature and VPX. On the side and the edge, rather large 95% Confidence Intervals occur, which further confirms that conditions in these zones are affected by the exact positions of the arms.

Section 5.2.1 highlighs that movement in the bed represents a confounding factor when comparing the field data with the Lectus assumptions for the boundary conditions. In order to separate the "movement effect" from other possible differences between the Lectus assumptions and the field, a 'still test' was carried out, where 6 participants were asked to stay still in Series 2 beds, for at least 20 minutes. The results of the 'still test' were described in this section, and they highlight that even by eliminating the effect of movement, there are still some differences between participants on the temperature and VPX recorded in the mattress' surface. However, the data analysis also revealed that the results from the 'still test' favourably compare *on average* with the Lectus assumptions, apart from some locations - particularly for the temperature in the sides, edges and feet

area. Some of these discrepancies are likely to be determined by the exact location of body parts on the mattress, which appear to particularly affect the temperature[6]. It can be concluded that if the confounding factor of movement is excluded, the Lectus assumptions favourably compare with field data, apart from the edges. However, the main aim of section 5.2 was to assess whether the Lectus assumptions favourably compare with results from the field, i.e. from real beds. The 'still test' does not necessarily reproduce "realistic conditions". Consequently, the next section aims to assess how different the results from the 'still test' are from the field data (Series 2).

5.2.3 Comparison of all results

In the previous section the Lectus boundary conditions were compared with the 'still test' data (bed surface locations only) carried out by 6 participants in three of the four Series 2 beds. The results highlighted that *on average* the conditions registered during the 'still test' favourably compare with the Lectus assumptions - apart from some locations (edges, sides, legs), particularly for the temperature. The 'still test', however, does not necessarily reproduce realistic conditions. This section firstly aims to assess how different the results from the 'still test' are, from the field data. Secondly, this section also aims to compare all the results analysed so far:

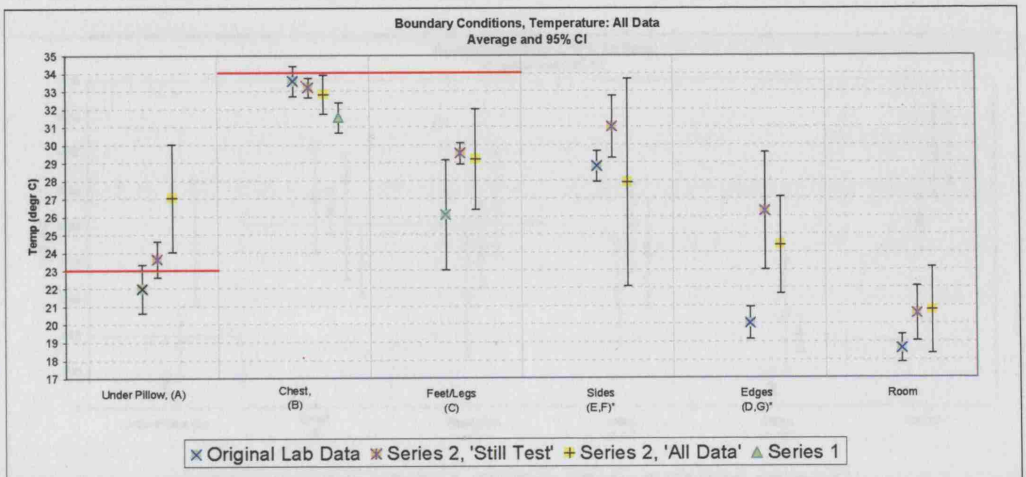
1. Lab data: 10 volunteers sleeping for 8 hours one at a time in the same single bed;
2. 'Still test': 6 volunteers lying still for at least 20 minutes in three Series 2 beds (same mattress, different sizes, different duvet and pillow, additional padded cover for one bed);
3. Series 2 data: 4 volunteers (3 of which also carried out the 'still test') monitored for 6 weeks in Series 2 beds (same mattress, different sizes, different duvet and pillow, additional padded cover for one bed);

[6] This also implies that more sensors should be used than those used in the field study, in order to build a better picture of the mattress boundary conditions.

4. Series 1 data: 8 volunteers, monitored for 6 weeks in different beds (under the chest area only).

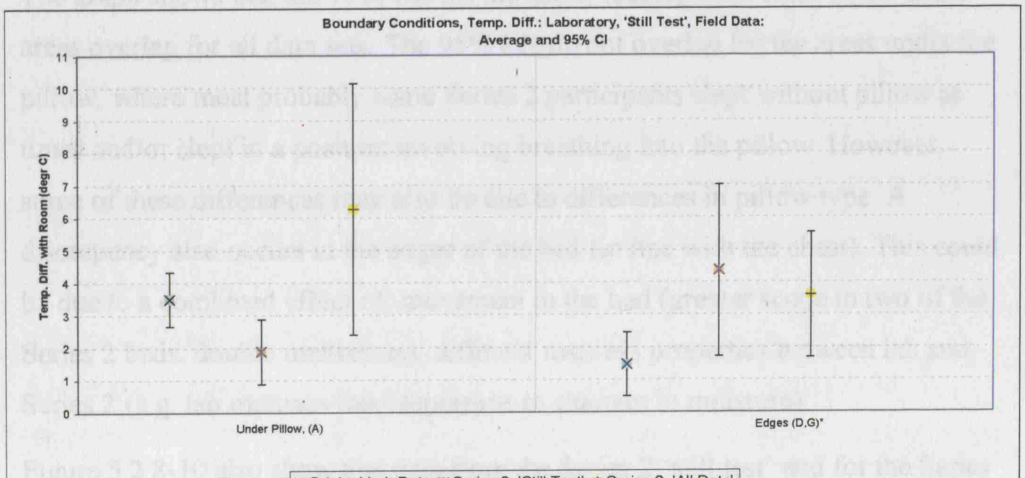
Figure 5.2.8 illustrates the average and 95% CI temperature (occupied bed) for each zone of the mattress surface, calculated for each of the data-sets. The graph shows that for the temperature under the chest, the 95% CIs overlap, apart from the data from Series 1. Due to the confounding factor of movement, it is not possible to establish whether the temperature in Series 1 is lower because of movement[7], or because of the different hygrothermal properties of Series 1 mattresses. The 95% CIs for the temperature in the feet/legs area, and on the sides of the chest also overlap – although the sides have a rather large 95% CI for the data from Series 2. Conditions under the pillow and on the edges (side of the chest) do not overlap fully. However, these conditions are the most affected by room conditions, and it may therefore be more meaningful to compare the results in terms of difference with room temperature. If conditions under the pillow and on the edges are compared in terms of difference with room temperature (Figure 5.2.9), the 95% CIs overlap - although there is a large 95% CI for Series 2, probably due to one of the participants sleeping without pillow at times.

[7] As previously mentioned, all mattresses in Series 1 were double beds, mostly occupied by 1 person, with a larger scope for movement when compared with the Series 2 beds (2 singles and 2 doubles, one of which occupied by 2 people).



Red Line: Lectus Assumptions.
*Average of the two locations (E,F; D,G). These locations are assumed to have the room temperature in Lectus.

Fig 5.2.8 Comparison of average and 95% CIs temperatures on the surface of the bed (occupied bed), for: lab data, 'still test', Series 2 data and Series 1 data (see Fig. 4.2.4 for reference of locations).



* Average of the two locations (D,G), see Fig. 4.2.2 for reference of locations

Fig 5.2.9 Comparison of average and 95% CIs temperature difference with room conditions on the surface of the bed (under pillow and edges, occupied bed), for: lab data, 'still test', Series 2 data.

Figure 5.2.10 illustrates the average and 95% CI VPXs (occupied bed) for each zone of the mattress surface, calculated for each of the data-sets.

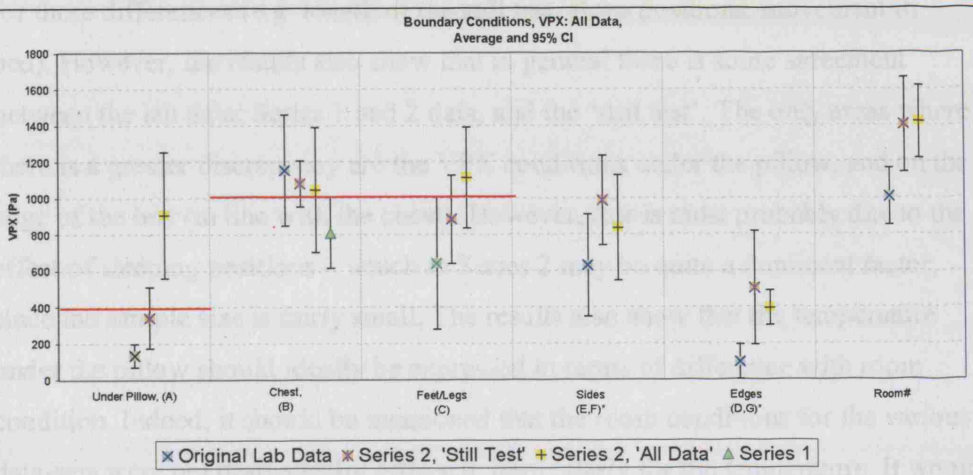


Fig 5.2.10 Comparison of average and 95% CIs VPXs on the surface of the bed (occupied bed), for: lab data, 'still test', Series 2 data and Series 1 data (see Fig. 4.2.4 for reference of locations).

The graph shows that the 95% CIs for the chest, feet/legs and sides of the chest areas overlap for all data sets. The 95% CIs do not overlap for the areas under the pillow, where most probably some Series 2 participants slept without pillow at times and/or slept in a position involving breathing into the pillow. However, some of these differences may also be due to differences in pillow type. A discrepancy also occurs in the edges of the bed (in line with the chest). This could be due to a combined effect of: movement in the bed (greater scope in two of the Series 2 beds, double mattresses); different mattress properties between lab and Series 2 (e.g. lab mattress less responsive to changes in moisture).

Figure 5.2.8-10 also show that data from the Series 2 'still test' and for the Series 2 'standard test' (referred to as 'all data' in the graph) mostly overlap, suggesting that on average the conditions between the two tests are not dissimilar – apart from conditions under the pillow, which is affected by sleeping positions (e.g. no pillow used during sleep).

This section compared the results from the 'still test' with the correspondent results from the field. The section also aimed to compare the results from all the data sets considered in this study. The results show that although some differences

exist between the 'still test' and field data (Series 2), differences in experimental set ups amongst the monitored bed make it difficult to establish the main reason for these differences (e.g. length of the still test, sleep positions, movement in bed). However, the results also show that in general there is some agreement between the lab data, Series 1 and 2 data, and the 'still test'. The only areas where there is a greater discrepancy are the VPX conditions under the pillow, and on the edge of the bed (in line with the chest). However, this is most probably due to the effect of sleeping positions – which in Series 2 may be quite a dominant factor, since the sample size is fairly small. The results also show that the temperature under the pillow should ideally be expressed in terms of difference with room condition. Indeed, it should be mentioned that the room conditions for the various data-sets were not dramatically different, particularly for the temperature. It would be interesting to repeat the experiments in the summer time, and check if the Lectus assumptions are valid (on average).

5.2.6 Boundary conditions: summary

Section 5.2 compared the Lectus boundary conditions with the fieldwork data. The results show that there is in general an agreement between predicted and monitored results *on average*. However, the results also show that there is a degree of variability in hygrothermal conditions on the bed surface, both within individuals and across individuals. This variability is due to a combination of: differences in heat and moisture output during sleep across individuals, as well as within individuals (e.g. moisture intake before sleep or different body temperatures during sleep cycles); clothing levels; different hygrothermal properties of mattresses, duvets and pillows; sleeping position (particularly for the head); movement levels during sleep (also affected by mattress size). Consequently, although *on average* the boundary conditions assumed in Lectus are sufficiently representative of fieldwork data, a *range* of conditions are likely to occur in reality. It is therefore necessary to establish whether the impact of such average hygrothermal conditions on house dust mite populations significantly differs from the impact of the extremes in the range. Consequently, the mite

population model Popmite should be used to assess the impact of 3 scenarios, summarised in Table 5.2.6 (see Chapter 12):

1. Boundary conditions as currently assumed in Lectus;
2. Boundary conditions corresponding to the "worst case scenario" (i.e. best for mite growth), i.e. highest temperature and highest VPX;
3. Boundary conditions corresponding to the "best case scenario", i.e. lowest temperature and lower VPX.

The ranges in Table 5.2.6 were selected from the 95% CIs of the lab data. This is simply because the 95% CIs from the Series 1 or Series 2 data-sets may be misleading, due to the confounding factor of single/double mattresses (i.e. more movement in a double mattress, whilst Lectus models single mattresses). A greater 95% CI was chosen for the VPX conditions under the pillow (150 Pa), since the field data suggests that in some cases this may be significantly higher.

It is necessary to establish whether this variability in boundary conditions may significantly affect the prediction in mite populations. This issue will be addressed in Chapter 12.

Table 5.2.6 Scenarios to be tested in Popmite, in order to establish the impact on mite populations of the range of hygrothermal conditions found in the field.

Bed Surf. Loc. (ref Fig 4.2.7)	Scenario 1 (Lectus)		Scenario 2 ("worst")		Scenario 3 ("best")	
	Temp. (°C)	VPX (Pa)	Temp. (°C)	VPX (Pa)	Temp. (°C)	VPX (Pa)
Under Pillow (A)	23	400	24	550	22	200
Chest (B)	34	1000	35	1300	33	700
Legs/Feet (C)	34	1000	35	1300	31	640
Side (E,F)	28	800	29	1080	26	520
Edge (D,G)	(0)*	0	(4)*	100	(0)*	(0)*

* Temperature difference between bed and room.

5.3 Changes in hygrothermal conditions with bed occupancy

Based on lab experiments with 8 participants sleeping one at a time in the same lab-bed, the Lectus model assumes that upon occupation each zone of the mattress surface reaches its final boundary conditions immediately, maintaining such conditions until the occupant leaves the bed. Once the occupant leaves the bed, it is assumed that the bed surface conditions gradually go back to equilibrium with the room conditions, following the formula:

$$T\_Bed^{n}=T\_Bed^{n-1}-\{[\Delta T^{n-1}]x[1-(0.5)^{(t/k)}]\} \quad [5.3.1]$$

where:

- T_Bed^{n} is the bed temperature at the time interval n;
- T_Bed^{n-1} is the bed temperature at the time interval (n-1);
- ΔT^{n-1} is the temperature difference between the bed and the room, at the time interval (n-1);
- n is the time interval in minutes;
- t is the time step in which room conditions are available in minutes (e.g. every 60 minutes);
- k is a constant, corresponding to the time it takes for the ΔT^{n-1} to be halved.

In Lectus, it is assumed that ΔT^{n-1} is halved every hour (k=60 min).

The same formula applies in Lectus for the decay of the vapour pressure.

In this section, the above assumptions are tested against fieldwork data.

It is necessary to know the exact times when the fieldwork participant got into bed and left the bed, in order to be able to accurately calculate the amount of time it took for the hygrothermal conditions to rise, and to drop back to room conditions. In the 'still test' - performed during the 'Series 2' study - the exact bed occupancy times were recored. The 'still test' was described in Section 5.2.2, where 'Series 2' participants were asked to lie in their bed for at least 20 minutes, keeping as still as possible. The hygrothermal conditions were logged minute by minute during each test.

In Lectus it is assumed that upon occupation, each zone of the mattress surface reaches its final boundary conditions immediately. This is of course an approximation of reality, as Figure 5.3.1 shows. However, since Lectus is usually run with hourly intervals, this assumption is reasonable, if it takes less than 1 hour for the bed surface to reach the final steady-state conditions upon bed occupancy. The 'still test' suggests that this may be the case, although a longer 'still test' would be required in order to prove this without any doubt.

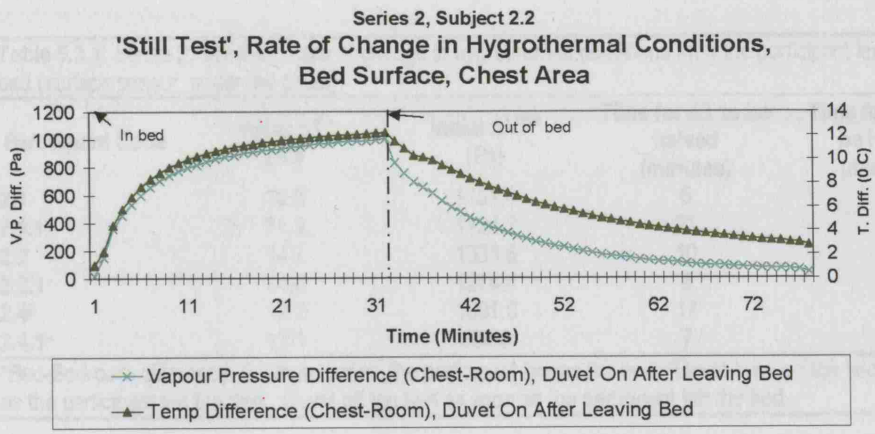


Fig 5.3.1 Series 2, 'still test', rate of change in hygrothermal conditions on the bed surface (chest area).

In Lectus it is also assumed that once the occupier leaves the bed, after one hour the ΔT (temperature difference) and the ΔVP (vapour pressure difference) between the bed and the room will be halved. Table 5.3.1 shows the time it took for the ΔT and the ΔVP to be halved, in the Series 2 'still test' study (bed surface, chest area). When considering the results, it should be taken into account that participant 2.1 and 2.1.1 performed the 'still test' on the same bed; similarly 2.2 and 2.2.1 used the same bed; 2.4 and 2.4.1 tested the same bed.

In the original Lectus lab experiments, participants were required to remove the duvet upon leaving the bed. During the 'still test' participant 2.4 left the duvet on the bed as soon as leaving the bed, whilst participant 2.4.1 pulled away the duvet when leaving the bed. The duvet position when leaving the bed was not recorded for the other participants, since the 'still test' was performed primarily to assess the effect of movement on boundary conditions.

Table 5.3.1: Series 2, 'still test'. Rate of change in hygrothermal conditions after the participant leaves the bed (surface sensor, under the chest)

Participant Code	Initial ΔT* (°C)	Initial ΔVP* (Pa)	Time for ΔT to be halved (minutes)	Time for ΔVP to be halved (minutes)
2.1	10.8	1151.4	5	1
2.1.1	11.2	1124.2	21	6
2.2	14.7	1033.6	10	2
2.2.1	14.8	1274.4	8	1
2.4#	12.2	1001.0	17	7
2.4.1~	12.1	808.8	7	3

*Bed-Bedroom difference, 1 minute before the participant leaves the bed; #Duvet back on the bed, as soon as the participant left the bed; ~Duvet off the bed as soon as the participant left the bed.

The differences amongst participants illustrated in Table 5.3.1 are likely to be due to: a) differences in duvet position when leaving the bed; b) differences in duvet type (in Series 2 the mattress only was standardised); c) differences in sensors' response times. Apart from these differences, the table shows that it takes at least twice as long for the temperature to go back to room conditions, than for the vapour pressure. The results also show that if the duvet covers the mattress immediately after the participant leaves the bed, it takes more than double the time for the ΔT and ΔVP to be halved, than when the bed is not made (participant 2.4 and 2.4.1). Moreover, in all cases it takes significantly less than 60 minutes for the initial ΔT and ΔVP to be halved.

When comparing these results with the Lectus assumptions, some differences between the Series 2 experimental set up and the original Lectus experimental set up should be highlighted:

- The mattress and the duvet used in Series 2 may have different hygrothermal properties than those used in the Lectus lab experiments. This would affect the rate of change in hygrothermal conditions.
- The 'still test' in Series 2 was only carried out for 20 minutes (30 minutes for participant 2.4 and 2.4.1), while the Lectus experiments for approximately 8 hours. This affects how deeply within the mattress the heat and moisture have travelled in the 'still test', which in turn would affect the rate at which the surface conditions change once the bed is unoccupied.

In order to check whether the length of time when the bed is occupied may affect the rate at which the hygrothermal conditions change after the bed is vacated, it

would be necessary to look at Series 2 data with recorded occupancy times. This type of data however is not available. Nonetheless, by analysing the plots of Series 2 data, it emerged that it was possible to clearly identify bed occupancy times for certain days corresponding to participant 2.1. This was the most "stationary" of the Series 2 participants, which makes it easier to dismiss temperature or VPX changes due to movements in bed.

By analysing the variation in hygrothermal conditions for bed 2.1, it emerges that after the participant leaves the bed, it takes approximately 20 to 40 minutes for the temperature difference between bed and room to be halved, and approximately 10-30 minutes for the VPX to be halved. In some occasions, however, it takes even less than 10 minutes (the logging interval in Series 2 was 10 minutes) for the hygrothermal conditions to be halved. This variability is probably dependent on whether (and for how long) the duvet is left off the bed, after the bed is vacated. By analysing the plots of bed 2.1 it also appears that it takes approximately 30-40 minutes for the temperature and the VPX to reach steady state conditions, which agrees with the Lectus assumption. It is however difficult to assess to what extent these results are confounded by participant 2.1 moving in bed. These results also suggest that the length of the 'still test' may have not been fully sufficient for the beds to reach steady state conditions, although participants 2.4 and 2.4.1 did carry out the 'still test' for 30 minutes, which may have been enough time.

This section aimed to establish whether the current Lectus assumptions on the rate of change in the boundary hygrothermal conditions once the occupier gets into/leaves the bed agree with the Series 2 fieldwork data. The results suggest that once the bed is vacated, bed conditions may return in equilibrium with room conditions more quickly in the field, than the rate assumed in Lectus. Furthermore, the fieldwork data suggests that the vapour pressure returns to room conditions more quickly than the temperature, whilst in Lectus it is assumed that temperature and vapour pressure return to room conditions at the same rate. The position of the duvet does affect the rate at which bed conditions go back to room conditions. On average, it could be concluded that the temperature difference between the bed and the room is halved in 30 minutes (half of what is currently assumed in Lectus), and the VPX is halved in 20 minutes (one third of what is currently assumed in Lectus). If the duvet covers the mattress immediately after

the bed is vacated, it takes more than double the time for the ΔT and ΔVP to be halved, than when the bed is not made

However, it should be highlighted that differences in experimental set ups between the fieldwork data and the Lectus lab-experiments (e.g. different mattress and duvet, different duvet position) may account for some of the discrepancies between Lectus predictions and field data. Furthermore, the Lectus model is not very sensitive to this input parameter (see Chapter 9).

5.4 Measured versus predicted conditions within the depth of the mattress

In Lectus the boundary conditions are used as input for a 3-dimensional transient heat conduction and vapour diffusion model, which is used to calculate the hygrothermal conditions at the centre of each mattress cell (see Chapter 4). This section compares the fieldwork results (Series 2) measured within the depths of the mattress with the Lectus predictions. In particular, the following sub-sections illustrate:

- The inputs and assumptions used for the initial model predictions (base-case) (5.4.1);
- A comparison between monitored and predicted base-case results (5.4.2);
- Assessment of the model uncertainties and their impact on the model's predictions (5.4.3);
- A summary discussion of the model's predictions for the hygrothermal conditions within the mattress (5.4.4).

5.4.1 Initial assumptions: base-case study

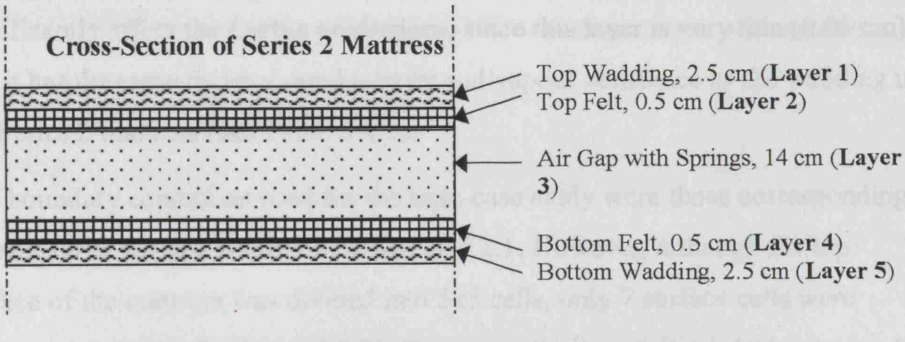
This section describes the inputs and assumptions used for the model predictions of a Series 2 mattress. In particular, it was decided to simulate the conditions found in mattress 2.1, which was one of the two single mattresses in Series 2. This

is because Lectus was developed to simulate single mattresses. Furthermore, only one half of the Series 2 *double* beds was monitored.

The main inputs of the Lectus model are:

1. The mattress geometry (i.e. number of layers and cells, with respective dimensions);
2. The room and the boundary conditions;
3. The physical properties of the mattress materials (density; thermal conductivity; specific heat capacity; vapour diffusion coefficient; specific moisture capacity).

The dimensions used in the base-case study were those of the Series 2 study mattress (single mattress), which was 0.91 m wide, 1.92 m long, and 0.2 m high. The mattress used in the Series 2 study was simulated by considering 5 layers, as illustrated in Figure 5.4.1. These layers reflected the actual layers found in the monitored mattress. Each layer was divided into 5x5 cells, which reflected the 5x5 division in the top mattress surface, for the boundary conditions. A total of 125 cells was therefore simulated. It was found that doubling the cells number would produce results within +/- 1% of the case with 125 cells. This was considered acceptable.



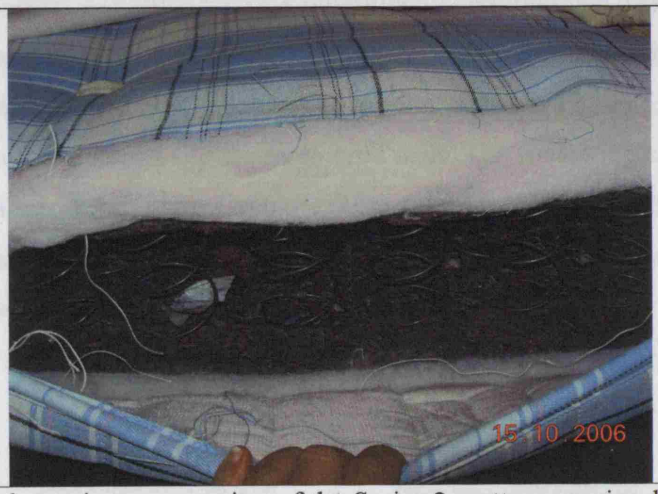


Fig 5.4.1 Schematic cross-section of the Series 2 mattress as simulated in Lectus, and picture of the inside of the mattress.

It should be mentioned that the Series 2 mattress's outer layer was cotton, whose hygrothermal properties were known (except the heat capacity). However, this thin layer was not included in the simulations, since its properties made the calculations unstable. In Lectus the stability criterion is given by:

$$\Delta t < \Delta\sigma = \frac{C_i}{\sum_j \frac{1}{R_{ij}}} \qquad [5.4.1]$$

where $\Delta\sigma$ is the stability criterion, in seconds; Δt is the calculations time-step, in seconds; $C_i=\rho_i C_i V_i$ (ρ=density, $kg.m^{-3}$; C=specific heat capacity, $J.kg^{-1}.K^{-1}$; V=volume, m^3); R is the thermal resistance, $K.W^{-1}$.

The temperature stability criterion obtained when the cotton layer was included (i.e. 7 layers overall) was 0.44 seconds. Since it was not possible to use a time

step calculation smaller than 0.5 seconds in Lectus, it was decided to eliminate the cotton layer. It was considered that the elimination of the cotton layer would not significantly affect the Lectus predictions, since this layer is very thin (0.05 cm), and it has the same thermal conductivity and vapour resistance as the wadding in the Series 2 mattress (see Table 5.4.1).

The boundary conditions used for the base-case study were those corresponding to the *measured* surface conditions of the Bed 2.1. However, although the top surface of the mattress was divided into 5x5 cells, only 7 surface cells were monitored in Series 2 (Figure 5.2.7). Consequently, assumptions had to be made on the boundary conditions in the remaining cells, since no other information was available to the author. These assumptions are illustrated in Figure 5.4.2, where the 7 white areas correspond to the monitored cells. The remaining cells (grey areas in the Figure 5.4.2) were given the same values of adjacent cells, or average values of cells on either side. The boundary conditions for the bottom and sides of the mattress were given the same conditions as the room.

Series 2 Mattress (Top Surface View)

=Av.(A,D)	=A	Under Pillow: A	=A	=Av.(A,D)
=D	Side: F	Chest: B	Side: E	Edge: D
=H	=Av. (B,C,H)	= Av.(B,C)	=Av. (B,C,H)	=H
=H	=Av. (H,C)	Legs/Feet: C	=Av. (H,C)	=H
Corner: H	=Av. (H,C)	=Av. (H,C)	=Av. (H,C)	=H

Fig 5.4.2 Boundary conditions for the top mattress surface utilised in Lectus to simulate bed 2.1, from the Series 2 study. The white cells correspond to the locations which were monitored in the field study (centre of the cell).

The other inputs for Lectus are the materials properties for each layer. As previously illustrated in Figure 5.4.1, the modelled Series 2 mattress consisted of:

1 Wadding (2.5 cm, layer 1 and 5);

2 Felt (0.5 cm, layer 2 and 4);

3 Air cavity with metallic springs (14 cm, layer 3).

The physical properties of the wadding and the felt which were required for Lectus were measured by Mr David Brook, from the *Performance Clothing Research Group*, at the *Centre for Technical Textiles*, University of Leeds. However, the specific heat capacity could not be measured, since no adequate test facilities were available at the Leeds Centre for Technical Textiles. The properties are illustrated in Table 5.4.1 and 5.4.2 (including the cotton cover, which was not simulated in Lectus). These properties were then converted at UCL into the appropriate units required by the Lectus model, resulting in the values illustrated in Table 5.4.3.

Table 5.4.1 Thermal insulation, water vapour transmission and density of Series 2 bed materials, as measured by the University of Leeds.

Sample	Thermal Insulation *BS4745 / ISO 5085-1 (Togs)	Water vapour transmission **BS7209 (%)	Density of sample at 105°C, gm/ cm³
Cover	0.13	99 (0.7 Ret)	0.169
Wadding	6.53	74 (11.6 Ret)	0.025
Felt	2.84	80 (8.0 Ret)	0.070

* Togmeter method; **Wet cup method; 10 Togs = 1 K m² / Watt; Ret Unit = m² Pa / Watt

Table 5.4.2 Water vapour regain of Series 2 bed materials based on BS1051, as measured by the University of Leeds.

Sample	Percentage Regain (at 20 °C)				
	30% RH	50% RH	100% RH	50% RH	30% RH
Cover	2.0	4.0	14.6	5.1	2.6
Wadding	0.84	1.0	1.25	1.1	0.91
Felt	2.4	3.5	7.9	4.2	3.7

Table 5.4.3 Properties of Series 2 mattress materials, as used in Lectus.

	Density ($kg.m^{-3}$)	Thermal conductivity ($W.m^{-1}.K^{-1}$)	Vapour permeability ($kg.m^{-1}s^{-1}Pa^{-1}$)	Specific moisture capacity ($kg.kg^{-1}.Pa^{-1}$)
Top Cover	169	0.04	2.93E-10	5.00E-05
Wadding	25	0.04	8.86E-10	6.00E-06
Felt	70	0.02	2.57E-10	3.00E-05

Since the specific heat capacity for the mattress materials was not measured, this was assumed to be 850 $J.kg^{-1}.K^{-1}$ (from the WUFI 2.2 database: Kunzel HM, 1999). This is the specific heat capacity of mineral wool, which was considered a similar material (similar density and thermal conductivity) to the wadding and the felt. The impact of the uncertainty in this input parameter will be in Chapter 9.

Assumptions also had to be made on the properties of the air gap in the sprung mattress. In previous work the properties of the air cavity had been assumed the same as still air (Cunningham *et al.*, 2004). However, in an air cavity the relative contributions of heat conduction, convection and radiation depend on: the surfaces' emissivity, the dimensions of the air cavity, the direction of the heat flow (horizontal/vertical), and the temperature difference between the two surfaces (CIBSE, 1986). Although many models - including Lectus - do not simulate convection and radiation *explicitly*, the additional transport mechanisms (convection, radiation) in an air cavity are often indirectly included in the thermal and diffusion resistance properties of the air cavity (CIBSE, 1986). Although information is usually available on the hygrothermal properties of air cavities (horizontal or vertical), the literature search did not reveal such information for an air cavity as thick as the one found in the mattress (14 cm). Figure 5.4.3 and 5.4.4 show the relationship between the thickness of an air cavity, and respectively: its thermal conductivity and its vapour resistance factor. The figures shown in the graphs were taken from the WUFI 2.2 materials database, which include the effect of convection and radiation for conditions usually found in buildings (Kunzel HM, 1999).

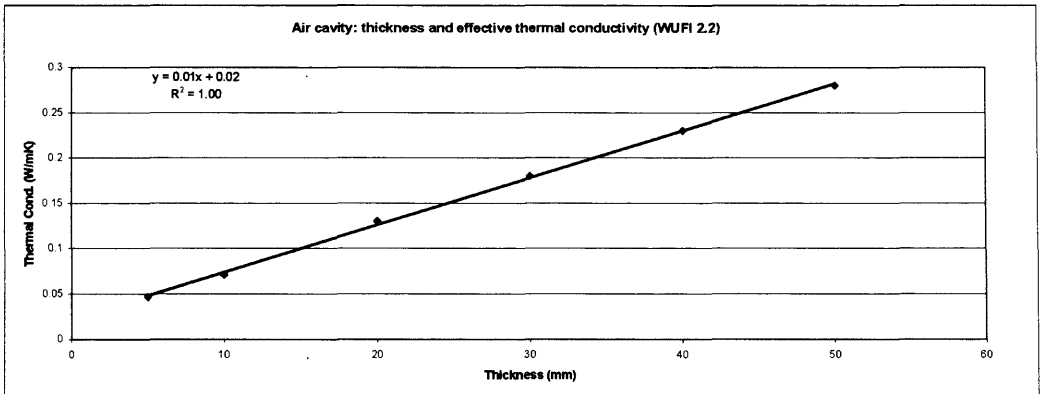


Fig 5.4.3 Relationship between the thickness of an air cavity, and its effective thermal conductivity (figures from WUFI 2.2, Kunzel HM, 1999).

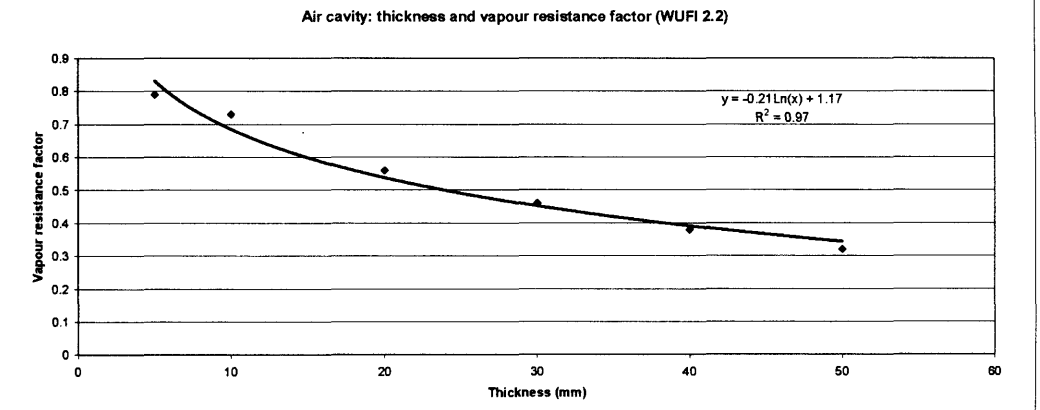


Fig 5.4.4 Relationship between the thickness of an air cavity, and its vapour resistance factor (figures from WUFI 2.2, Kunzel HM, 1999).

The graphs show that there is a clear relationship between the air cavity's thickness, and its properties. Consequently, the assumption was made that it was reasonable to extrapolate the thermal conductivity and the vapour resistance factor, based on the available data. The thermal conductivity for the 14 cm air cavity was therefore assumed as 0.75 W/mK, and the vapour resistance factor was assumed as 0.1. This corresponds to a vapour permeability of 2.02E-09 $kg.m^{-1}s^{-1}Pa^{-1}$ - since the vapour resistance factor is the ratio between the vapour permeability of stagnant air[9] and that of the material under identical conditions (Kumaran, 1996). It should be mentioned that a certain level of uncertainty is associated with these chosen values. For example, the air cavity in the mattress is

[9] The water vapour permeability of air is equal to $[2x(10^{-7}) \times (T^{0.81})] \times (Pl^{-1})$, where T is the temperature in Kelvin and Pl is ambient atmospheric pressure in Pascal (WUFI 2.2). At 20 °C, and 101,325 Pa atmospheric pressure, the vapour diffusion coefficient of air is approximately 2×10^{-10} $kg.m^{-1}.s^{-1}.Pa^{-1}$.

not necessarily under the same conditions as the air cavities tested in Figure 5.4.3 and 5.4.4. Furthermore, the air cavity in the mattress has metallic springs, which also affect its properties. The impact of this uncertainty will be discussed in Chapter 9.

The density, specific heat capacity and specific moisture capacity of the air cavity were also taken from the WUFI 2.2 database. Table 5.4.4 summarises the properties of the air gap utilised in the base-case simulation.

Table 5.4.4 Properties of the air gap used for the base-case in Lectus

	Density ($kg.m^{-3}$)	Effective Thermal conductivity ($W.m^{-1}.K^{-1}$)	Specific heat capacity ($J.kg^{-1}.K^{-1}$)	Vapour permeability ($kg.m^{-1}s^{-1}Pa^{-1}$)	Specific moisture capacity ($kg.kg^{-1}.Pa^{-1}$)
Air cavity (14 cm)	1.3	0.75	1000	2.02E-09	0.000006

So far the assumptions utilised for simulating bed 2.1 were described. The next section illustrates the results, and compares them with measured values.

5.4.2 Base-case: results

Based on the assumptions illustrated in the previous section, an initial base-case scenario was run in Lectus for the initial monitored 48 hours. It was decided to focus the data analysis on the cells corresponding to the mattress area underneath the occupant's chest. This is for 2 main reasons:

- The area under the occupant's chest was the only surface cell where most neighbouring cells had been monitored as well (see Fig. 5.4.2, cell B);
- The area under the occupant's chest has the greater temperature gradient from the top to bottom of the mattress, when the bed is occupied. This means that there is greater scope for discrepancies between predicted and measured results.

Figure 5.4.5 shows a schematic representation of the mattress cross-section under the chest area, with the position of the sensors.

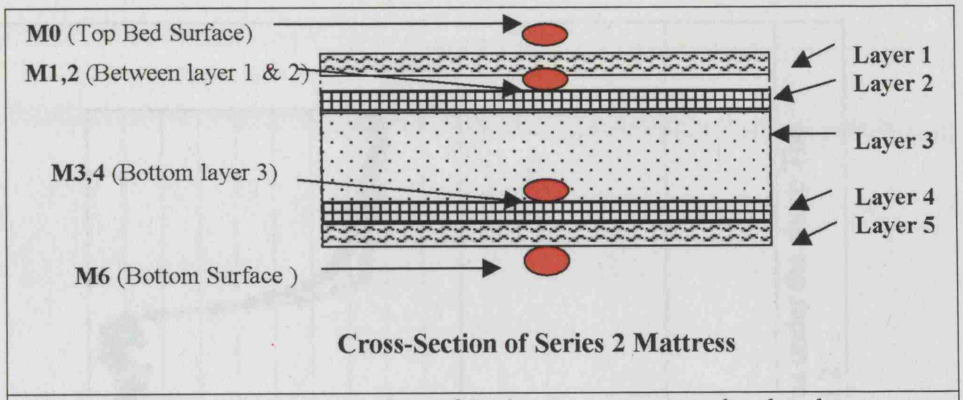


Fig 5.4.5 Schematic cross-section of Series 2 mattress, under the chest area. The red dots represent the sensors, whilst Lectus predicts conditions in the *middle* of each layer (layers indicated on the right).

Figure 5.4.6 (a and b) illustrates the predicted conditions over 48 hours in the Bed 2.1, in the top two layers (layer 1 and 2). These are compared with the measured conditions. In Series 2 the sensor was placed *between* layer 1 and 2, while Lectus is predicting the conditions in the *middle* of each layer. Consequently, the predictions for layer 1 and 2 were interpolated. It was assumed that a linear relationship between hygrothermal conditions and mattress depths could be adopted, particularly because layer 1 and 2 were rather thin. Figure 5.4.6 shows that predictions follow the changes in measured conditions rather well, and that the predicted temperatures are rather close to the measurements. However, there is an over-prediction in vapour pressures, when the bed is occupied. When the bed is not occupied, the mattress goes back to room conditions after a certain amount of time. Once this occurs, predictions and measurements are almost identical, since there is no difference in boundary conditions between the top and bottom of the mattress.

Figure 5.4.7 shows the Lectus predictions for the air gap, in comparison with the measured values. Again, although Lectus predicts the values in the *middle* of the air gap, in Series 2 the correspondent sensor was at the *bottom* of the air gap (see Figure 5.4.5). Consequently, the predicted values for the air gap correspond to an interpolation of the predictions for layer 3 (air gap) and of layer 4 (bottom felt). In Figure 5.4.7 it can be seen that the temperature predictions match adequately the measurements – although there is a little lag when the bed is just being occupied. However, there are some over-predictions in vapour pressures, when the bed is occupied.

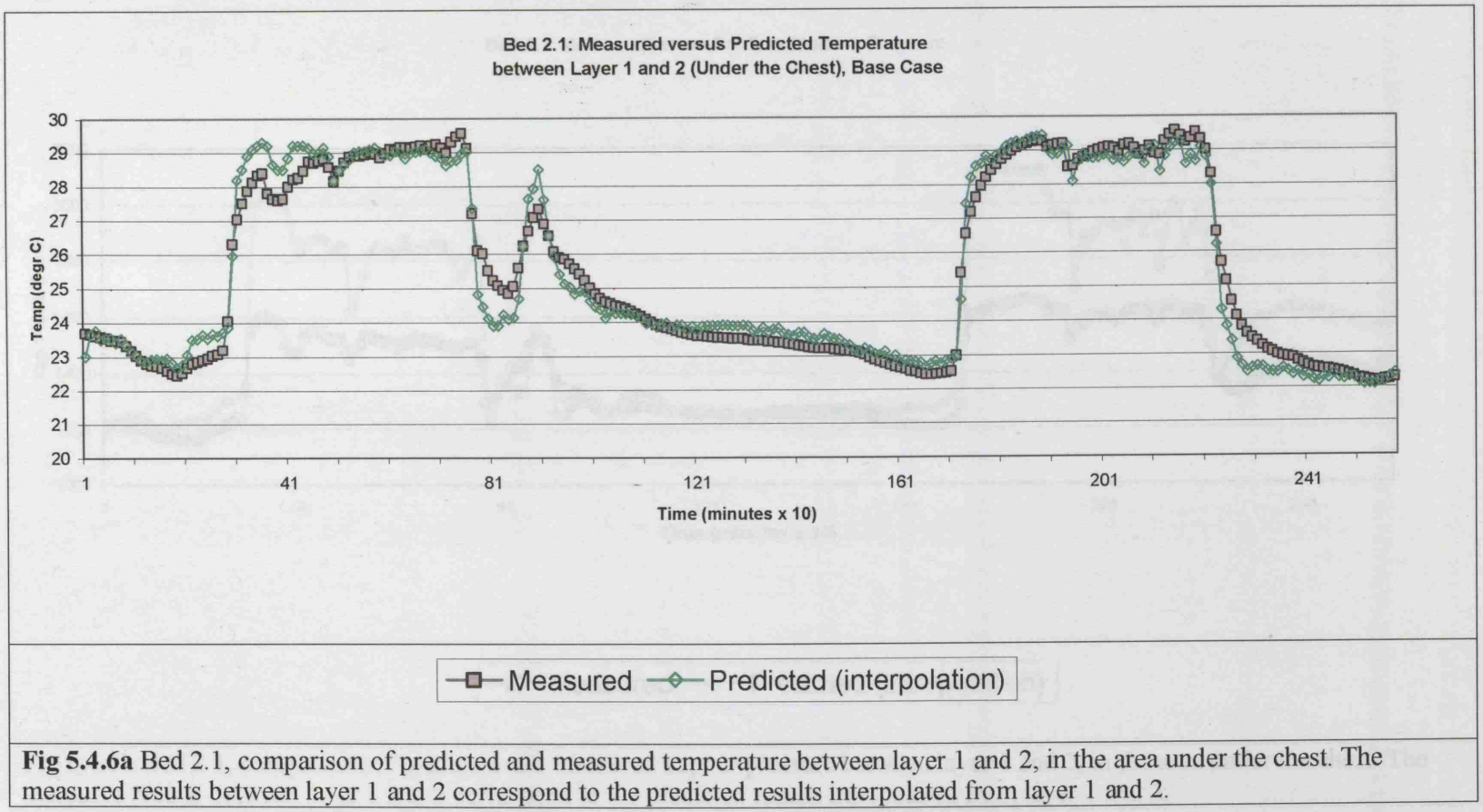


Fig 5.4.6a Bed 2.1, comparison of predicted and measured temperature between layer 1 and 2, in the area under the chest. The measured results between layer 1 and 2 correspond to the predicted results interpolated from layer 1 and 2.

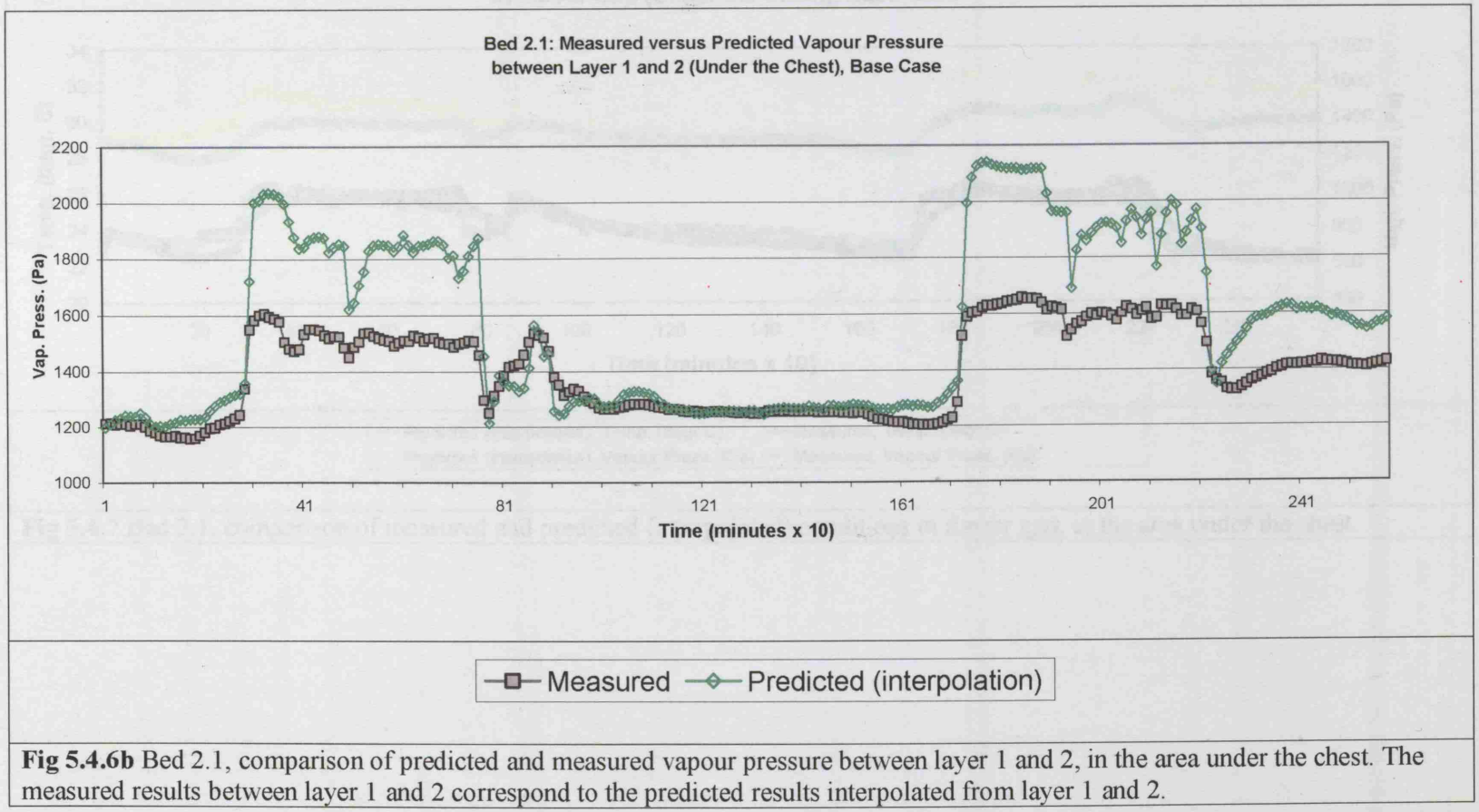


Fig 5.4.6b Bed 2.1, comparison of predicted and measured vapour pressure between layer 1 and 2, in the area under the chest. The measured results between layer 1 and 2 correspond to the predicted results interpolated from layer 1 and 2.

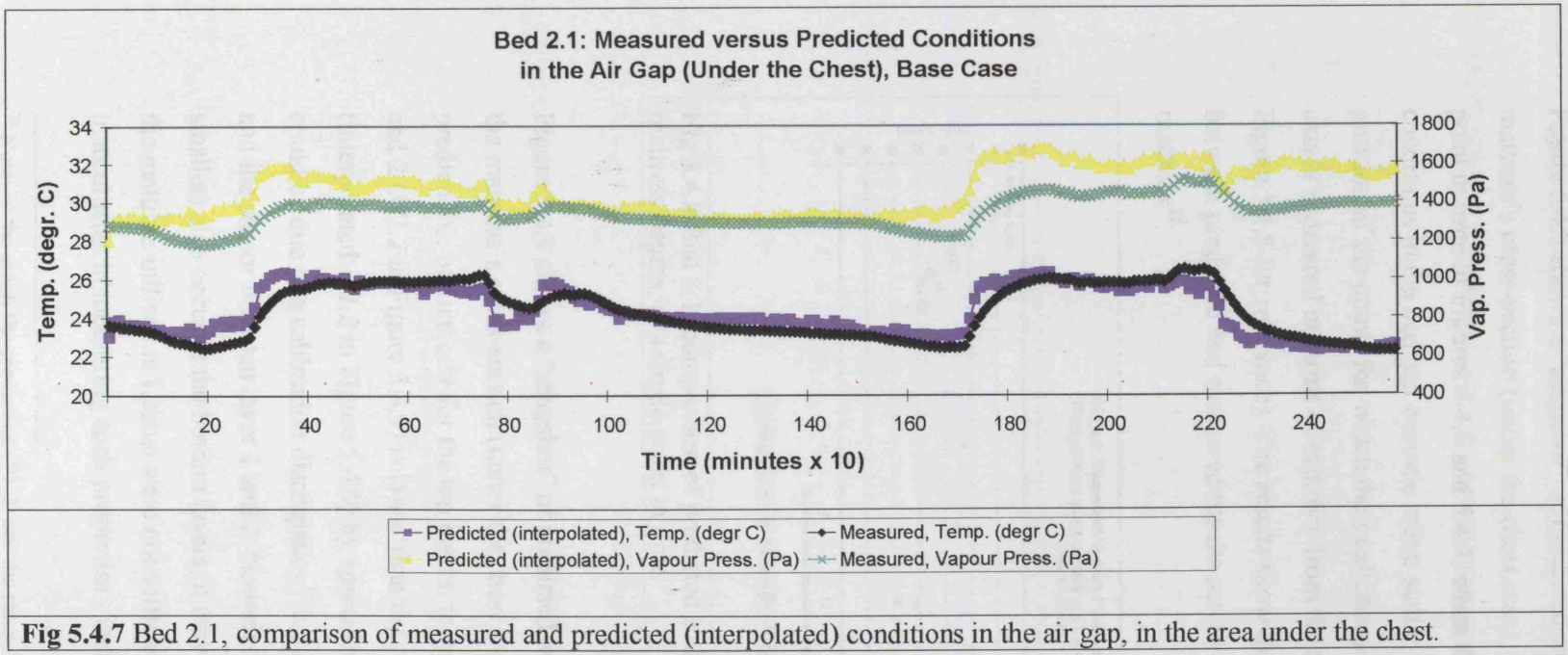


Fig 5.4.7 Bed 2.1, comparison of measured and predicted (interpolated) conditions in the air gap, in the area under the chest.

Figure 5.4.8 shows a "snapshot" of predicted and measured temperatures in the mattress's cross-section (under the chest area). This snapshot corresponds to a point in time of Figures 5.4.6 and 5.4.7, when the bed was occupied and the conditions in the bed had become rather stable. On the x-axis of Figure 5.4.8, the position of the points for which the predictions/measurements are given on the y-axis is expressed in terms of *distance* from the top surface of the mattress (see Figure 5.4.5 for reference). The results show that there is a good agreement between predicted and measured results across the various layers within the mattress[12].

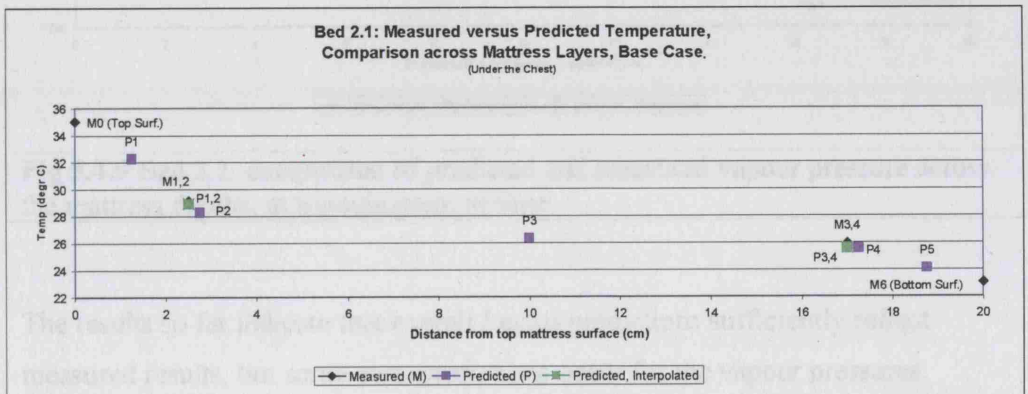


Fig 5.4.8 Bed 2.1, comparison of predicted and measured temperature across the mattress depths, at a single point in time.

Figure 5.4.9 shows a "snapshot" of predicted and measured vapour pressures in the mattress's cross-section (under the chest area). The results reveal over-predictions, particularly for the top layers: the measured values between layer 1 and 2 (*M1,2* in Figure 5.4.9) is lower than the corresponding predicted values (interpolated: *P1,2* in Figure 5.4.9) by approximately 300 Pa. This difference could be due to a calibration discrepancy[13] between the sensor on the top surface, and the sensor between layer 1 and 2. However, since over-predictions (although smaller) also occur in the bottom layers, it may be possible that the properties of the mattress utilised in Lectus were not sufficiently accurate. This could be due to inaccuracies in measuring such properties - including the impact of different test

[12] Note: in the graph the prediction (P1,2) and the measurement (M1,2,) for the mattress location between layer 1 and 2 overlap, therefore only one is visible.
[13] 300 Pa correspond to a 7.5% RH, for a temperature of 29 °C which was measured between layer 1 and 2.

conditions on the properties[14] - or to the effect of deformation when the mattress is occupied. These issues are discussed further in the following section.

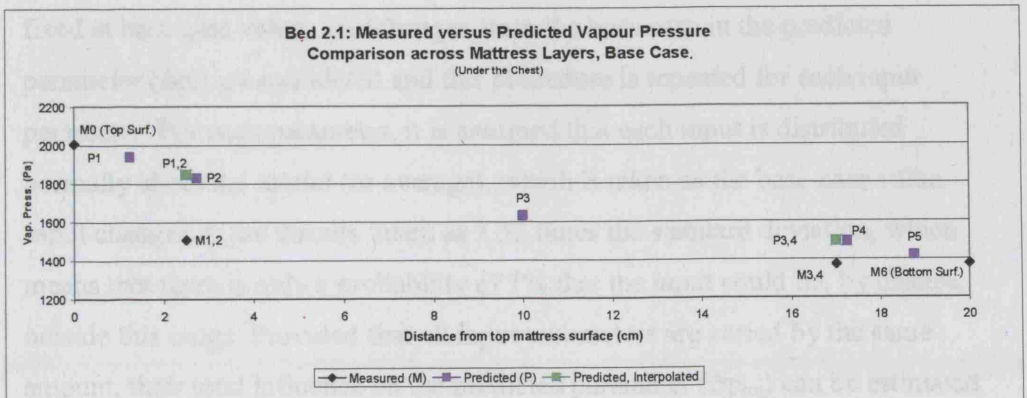


Fig 5.4.9 Bed 2.1, comparison of predicted and measured vapour pressure across the mattress depths, at a single point in time.

The results so far indicate that overall Lectus predictions sufficiently reflect measured results, but some over-predictions occur for the vapour pressures. However, the results described so far are based on a number of assumptions, to which a certain degree of uncertainty is associated. The impact of these uncertainties (including sensor's inaccuracies) on the model's predictions is discussed in the next section.

5.4.3 Impact of uncertainties on Lectus predictions

In the previous section the Lectus predictions for a base-case scenario were compared with the measured results. In general, the results indicated that overall Lectus predictions reflect measured results sufficiently. However, some over-predictions were encountered for the vapour pressures, particularly for the top layers of the mattress. This section estimates the impact of the model's uncertainties on its predictions, and establish whether the discrepancies between measurements and predictions can be attributed to such uncertainties.

[14] When the mattress is occupied, the hygrothermal conditions are different from typical test conditions.

In order to assess the impact of the model's uncertainties, the Differential Sensitivity Analysis method was utilised (Lomas and Eppel, 1992), which was reviewed in Chapter 2. Briefly, the Differential Sensitivity Analysis (DSA) involves varying one input variable *i* at a time, whilst the remaining inputs stay fixed at base case value. The changes from the base case in the predicted parameter (Δp_i) are calculated and this procedure is repeated for each input parameter. For each parameter, it is assumed that each input is distributed normally about the modal (or average), which is taken as the base-case value. Input changes Δi are usually taken as 2.33 times the standard deviation, which means that there is only a probability of 1% that the input could lie, by chance, outside this range. Provided that all input parameters are varied by the same amount, their total influence on the predicted parameter (Δp_{tot}) can be estimated as:

$$\Delta p_{tot} = \sqrt{\sum_i \Delta p_i^2} \qquad [5.4.1]$$

A list of all the uncertainties taken into account for the Lectus predictions is provided in Table 5.4.5, which also lists the input changes (Δi) utilised for each input parameter.

Table 5.4.5 Uncertainties considered for the Differential Sensitivity Analysis, and correspondent input changes Δi.

Input Parameter	Base Case Value	Input Change Δi (2.33xst. dev.)
Air Cavity, Effective Thermal Conductivity	0.75 W/mK	0.2 W/mK
Air Cavity, Vapour Resistance Factor	0.1	0.08
Wadding & Felt, Heat Capacity	850 J/kgK	500 J/kgK
Wadding, Density	25 kg/m^3	10% of base case
Wadding, Thermal Conductivity	0.04 W/mK	10% of base case
Wadding, Vapour Permeability	8.9E-10 kg/msPa	10% of base case
Wadding, Moisture Capacity	6.0E-06 kg/kgPa	10% of base case
Felt, Density	70 kg/m^3	10% of base case
Felt, Thermal Conductivity	0.02 W/mK	10% of base case
Felt, Vapour Permeability	2.6E-10 kg/msPa	10% of base case
Felt, Moisture Capacity	3.0E-05 kg/kgPa	10% of base case
Measurement Error for the Input Boundary Conditions under the Chest Area	(n.a.)	Temp.= 1 °C RH = 5%
(Measurement Error of the sensor between layer 1 and 2, against which the predictions are compared)	(n.a.)	Temp.= 1 °C RH = 5%

The DSA method requires knowledge of modal/average values for each input parameter, and their standard deviations. However, this is not always straightforward. For example, in the previous section the difficulties associated with estimating the effective thermal conductivity and vapour resistance factor for the air cavity in the sprung mattress were discussed. These difficulties are due to: a) no information was found on air cavities as large as the one found in the mattress (14 cm); b) the conditions to which the mattress air cavity is exposed might differ from standard test conditions; c) the metal springs in the air cavity will modify its hygrothermal performance. Due to lack of information, the author had to estimate the standard deviation for the properties of the air cavity. This was done by considering Figure 5.4.3 and 5.4.4, and by checking the properties of other materials. It was assumed that the other properties of the air cavity (density, moisture capacity and heat capacity) would have an insignificant uncertainty.

Another source of uncertainty was the heat capacity of the wadding and felt, which had not been measured. In the base case this was estimated as 850 J/kgK, which is the heat capacity of mineral wool (Kunzel HM, 1999). The input change Δi for the heat capacity was established by considering the heat capacities of other materials similar to wadding and felt. It was estimated that it was unlikely (less

than 1% chance) the heat capacity for the wadding and the felt could be higher than 1350 J/kg or lower than 350 J/kg.

The remaining properties of the wadding and the felt had been measured by the Centre for Technical Textiles, University of Leeds. However, these measurements are also subject to uncertainty. For example, a study found that the water vapour resistance of some building materials can be identified with a certainty of +/- 10% (Nordtest, 2003). It was therefore decided that the input change Δi for those input properties which had been measured would be 10% of the measured values.

When the mattress is being used, some deformation is likely to occur, especially in the top layers. This affects the mattress geometry as well as its hygrothermal properties (e.g. density). However, in order to assess the effect of the deformation, information about the extent of such deformation and its effects on the material's properties is needed. Since the deformation occurring in the mattress was not measured, and little information is available on the properties of mattress materials in general, the effect of deformation was not included in the Differential Sensitivity Analysis.

Another source of uncertainty in the model is related to the input boundary conditions, which were measured by using sensors with a specific accuracy[15]. The author tried to contact the sensors manufacturers, to obtain information about the average and standard distribution of the measurement error. However, the manufacturer's suggested the quoted accuracy should be used, which was therefore assumed equivalent to +/-2.33 x standard deviations. It should also be mentioned that the top surface of the modelled mattress is divided into 25 cells. However, it would have been far too lengthy to change the input boundary conditions for each cell, one at a time. Since the previous section (5.4.2) on the base-case results focused on the mattress section under the chest area, it was decided to vary the input boundary conditions of the surface cell corresponding to the chest area only.

Finally, the model's predictions for the mattress layers have to be compared with the measurements, which are also affected by a certain degree of error. Again, the

[15] For the temperature: Type K thermocouple (accuracy: 1 °C). For the RH, Honeywell HIH-3610 Series was used, which was calibrated by using Hobos H8 (accuracy: 5%).

manufacturer's quoted accuracies for the relevant sensors were utilised for the input change Δi.

In Figure 5.4.10 the dark blue points show the temperature difference between measured (between layer 1 and 2) and predicted (interpolated, layer 1 and 2) results, over a 48 hours period. The error band due to uncertainties associated with the input material properties is also plotted (pink points). Figure 5.4.11 is similar to 5.4.10, but the error band also *includes* the effect of error measurement (i.e. +/- 1 °C for the input boundary conditions and for the measurement results).

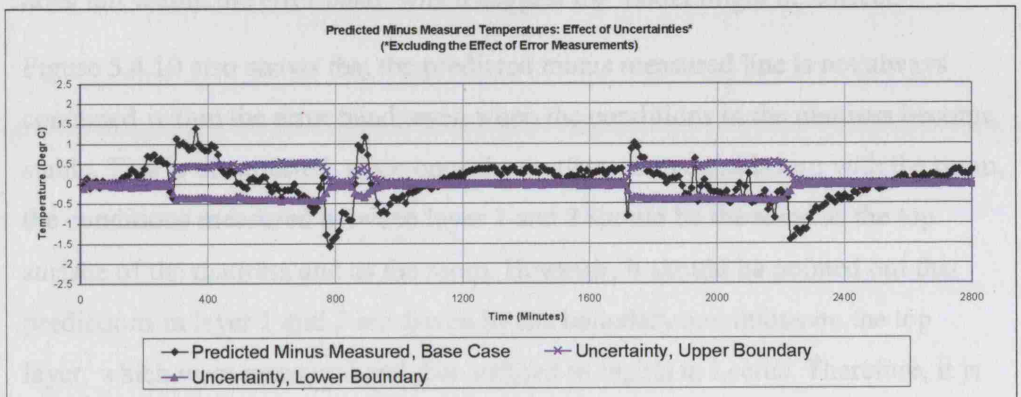


Fig 5.4.10 Predicted minus measured temperatures between layer 1 and 2 of the mattress: effect of uncertainties (*excluding* the effect of error measurement).

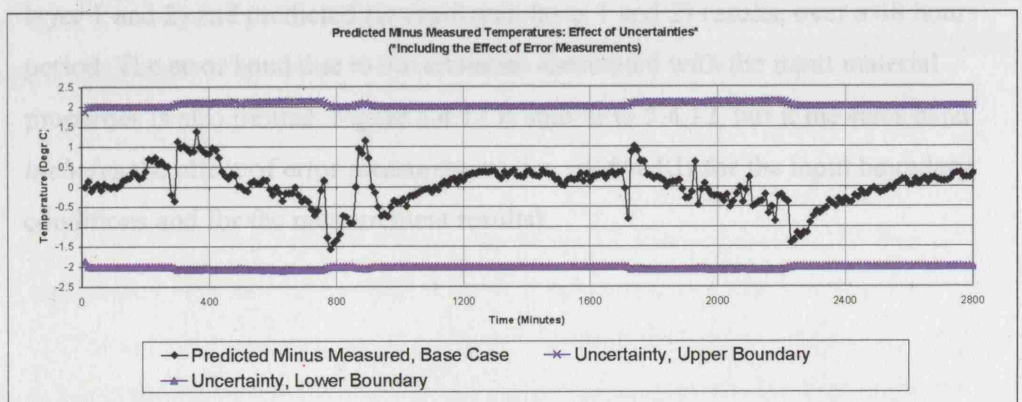


Fig 5.4.11 Predicted minus measured temperatures between layer 1 and 2 of the mattress: effect of uncertainties (*including* the effect of error measurement).

Figure 5.4.10 shows that when the bed is unoccupied and back to equilibrium with room conditions (in the graph this corresponds to sections where the upper and

lower uncertainty coincide), the effect of changes in materials' properties is nil: since there is no temperature gradient between the top and the bottom of the mattress, the various layers have the same temperature predictions as the base case – regardless of changes in thermal properties. However, when the bed is occupied, the effect of uncertainties in input hygrothermal properties is approximately +/- 0.5 °C. Figure 5.4.10 also shows that the predicted minus measured line is not always contained within the error band. However, once the effect of error measurement is taken into account (Fig 5.4.11), the predicted minus measured line does fall within the error band, which suggest the model might be correct.

Figure 5.4.10 also shows that the predicted minus measured line is not always contained within the error band, even when the conditions in the mattress become stable. This is unexpected, since once the mattress is in equilibrium with the room, the conditions measured between layer 1 and 2 should be the same as the top surface of the mattress and as the room. However, it should be pointed out that predictions in layer 1 and 2 are driven by the boundary conditions on the top layer, which were measured and then utilised as inputs in Lectus. Therefore, it is most likely that measurement discrepancies between the sensor on the top surface and the sensor between layer 1 and 2 are the cause of this incongruity.

Figure 5.4.12 shows the vapour pressure difference between measured (between layer 1 and 2) and predicted (interpolated, layer 1 and 2) results, over a 48 hours period. The error band due to uncertainties associated with the input material properties is also plotted. Figure 5.4.13 is similar to 5.4.12, but it the error band *includes* the effect of error measurement (i.e. +/- 5% RH for the input boundary conditions and for the measurement results).

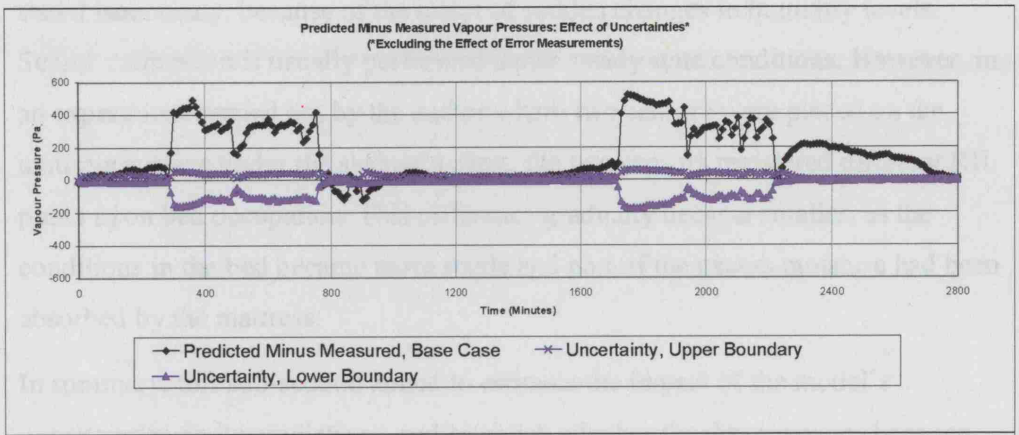


Fig 5.4.12 Predicted minus measured vapour pressures between layer 1 and 2 of the mattress: effect of uncertainties (*excluding* the effect of error measurement).

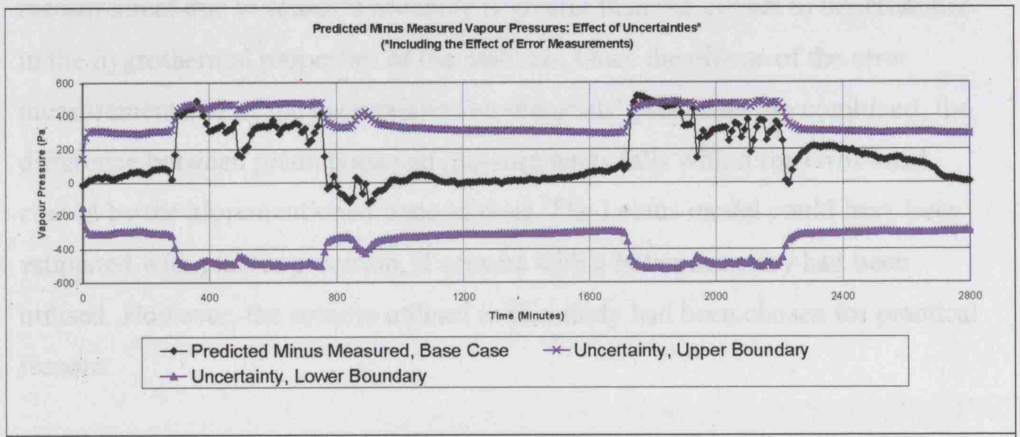


Fig 5.4.13 Predicted minus measured vapour pressures between layer 1 and 2 of the mattress: effect of uncertainties (*including* the effect of error measurement).

Similarly to the temperature graphs, Figure 5.4.12 shows that when the bed is unoccupied and back to equilibrium with room conditions, the error band (excluding error measurements) is nil, whilst when the bed is occupied the effect of uncertainties in input hygrothermal properties is up to 200 Pa. Figure 5.4.10 also shows that the predicted minus measured line is not always contained within the error band, particularly when the bed is occupied. However, once the effect of error measurement is taken into account (Fig 5.4.13), the predicted minus measured line mostly fall within the error band, except for very small peaks occurring when the bed is just being occupied. This could be, for example, because the surface sensor under the chest area (which determines the input

boundary conditions) tends to over-predict the RH more than the manufacturer-stated inaccuracy, because of the effect of sudden changes in humidity levels. Sensor calibration is usually performed under steady state conditions. However, in an experiment carried out by the author where two sensors were placed on the mattress surface under the sleeper's chest, the two sensors registered different RH peaks upon bed occupation. This difference gradually became smaller, as the conditions in the bed became more stable and part of the excess moisture had been absorbed by the mattress.

In summary, this sub-section aimed to estimate the impact of the model's uncertainties on its predictions, and establish whether the discrepancies between measurements and base-case predictions could be attributed to such uncertainties. The results of the Differential Sensitivity Analysis show that the impact of error measurement due to sensor's accuracy is greater than the impact of uncertainties in the hygrothermal properties of the mattress. Once the effects of the error measurement and of the uncertainties on materials' properties are combined, the difference between predictions and measurements falls within the error band caused by the aforementioned uncertainties. The Lectus model could have been estimated with greater precision, if sensors with a better accuracy had been utilised. However, the sensors utilised in this study had been chosen for practical reasons.

5.4.4 Predictions within the mattress: summary

Section 5.4 compared the fieldwork results (Series 2) measured within the depths of the mattress with the correspondent Lectus predictions.

The results of an initial base-case scenario indicated that overall Lectus predictions reflect measured results adequately, with some over-predictions for the vapour pressures. Once the effect of uncertainties on the model's predictions was estimated, it appeared that the impact of error measurement due to sensor's accuracy was greater than the impact of uncertainties in the hygrothermal properties of the mattress. If the effects of the error measurement and of the uncertainties on materials' properties are combined, the difference between

predictions and measurements falls within the error band caused by the aforementioned uncertainties.

The Lectus model could have been estimated with greater precision, if sensors with a better accuracy had been utilised. For future studies, it is recommended that the accuracy and the response time of such sensors are assessed under transient conditions, as opposed to steady-state ones.

In principle, a greater number of monitored cells would have allowed the comparison of predictions and measurements of a greater number of cells, increasing the confidence in the model's predictions. However, since the greater discrepancies between measurements and predictions occurred when the gradient between the mattress's top and bottom surfaces is larger, the measurement of the cells under the chest area – which was measured in the fieldwork and where such greater gradient occurs – is actually the most crucial for the model's assessment.

5.5 Summary discussion

This chapter discussed the validity of the Lectus model, by comparing its predictions with the fieldwork results (Series 1 and 2). The main elements constituting the Lectus model were addressed separately.

Firstly, the boundary conditions assumptions in Lectus were compared with the results from Series 1 and from Series 2, and from the original lab work which was carried out to develop the Lectus assumptions. The results show that *on average* the boundary conditions assumed in Lectus are sufficiently representative of fieldwork data. However, a *range* of conditions are likely to occur in reality, since there is a degree of variability in hygrothermal conditions on the bed surface, both within individuals and across individuals. It is therefore necessary to assess whether this variability may have a significant impact on mite populations. This variability is probably due to a combination of different factors, including differences in heat and moisture output during sleep within and across individuals; clothing levels; different hygrothermal properties of mattresses, duvets and pillows; movement levels during sleep, etc. The data analysis of the boundary conditions would have benefited by a greater number of surface sensors, and by recording the exact bed occupancy times. Standardisation of duvet and pillows

may also contribute in reducing the scope for differences across participants, thus aiding the comparison. Since the boundary conditions also depend on the mattress properties, they should be used with caution when attempting to use them to replicate the exact conditions in a specific bed whose properties are significantly different from those used for the fieldwork study.

This chapter also discussed the Lectus assumption on the rate of change in hygrothermal boundary conditions when the person gets into/leaves the bed. The data analysis revealed that the Lectus assumption of mattress conditions reaching steady-state values within one hour once the bed is occupied is reasonable. The data analysis also revealed that bed conditions go back to room values at a faster rate than what was originally assumed in Lectus, particularly for the vapour pressure. This however may be due to differences in mattress properties between the original lab-work and the fieldwork. The position of the duvet also appears important: if the duvet covers the mattress immediately after the bed is vacated, it takes more than double the time for the ΔT and ΔVP to be halved, than when the bed is not made. These differences may however not be crucial from the model's predictions and for the population model (see sensitivity analysis, Chapter 9).

Finally, this chapter compared the fieldwork results (Series 2) with the Lectus predictions of depth within the mattress. The results indicated that once the effect of uncertainties on the model's predictions was estimated, the impact of error measurement due to sensor's accuracy was greater than the impact of uncertainties in the hygrothermal properties of the mattress. If the effects of the error measurement and of the uncertainties on materials' properties are combined, the difference between predictions and measurements falls within the error band caused by the aforementioned uncertainties. The Lectus model could have been estimated with greater precision, if sensors with a better accuracy had been utilised. However, for future studies it is recommended that the accuracy and response time of such sensors are assessed under transient conditions, as opposed to steady-state only.

In summary, the data analysis showed that the Lectus predictions are *on average* comparable with fieldwork data. However, the bed simulated in Lectus is representative of average conditions with a "typical" sprung mattress. Lectus cannot - and has not been designed for - simulate specific mattresses with

complicated designs, nor it is fully representative of the variability of hygrothermal conditions occurring in real beds. In other words, worst-case scenarios or extreme conditions might occur in reality (e.g. high temperatures or sweating due to flu; mattress with an impermeable layer, etc.), which are not necessarily taken into account in Lectus. Lectus is however very useful to assess the *average* effect that room conditions might have on an *average* occupied bed.

Chapter 5: References

CIBSE, (1986), *CIBSE Guide, Volume A: Design Data*, London: CIBSE.

Cunningham MJ, Roos C, Gu L, Spolek G, (2004), Predicting psychrometric conditions in biocontaminant microenvironments with a microclimate heat and moisture transfer model - description and field comparison, *Indoor Air*, 14 (4): 235-242.

Fanger PO, (1970), *Thermal comfort: analysis and applications in environmental engineering*, New York: McGraw-Hill.

Kumaran, (1996), *IEA Annex 24. Heat, air and moisture transfer in insulated envelope parts. Final report. Volume 3. Task 3: Materials Properties*, Belgium: Laboratorium Bouwfysica.

Kunzel HM, (1999), *WUFI 2.2E: Simultaneous heat and moisture transfer in building components*. Fraunhofer Institute of Building Physics, Branch Institute Holzkirchen.

Lomas and Eppel, (1992), Sensitivity analysis techniques for building thermal simulation programs, *Energy and Buildings*, 19: 21-44.

Nordtest, (2003), *Intercomparison on measurement of water vapour permeance. Nordtest project agreement 1529-01. Poroject report 339-2003*, http://www.byggforsk.no/visVedlegg.aspx?vedleggID=261&dokumentID=1173, accessed: Oct 2006.

Oreszczyn T, Hong S, Ridley I, Wilkinson P, The Warmfront Study Group, (2006), Determinants of winter indoor temperatures in low income households in England, *Energy and Buildings*, 38: 245-252.

Pretlove SEC, Oreszczyn T, Ridley I, Wilkinson T, Crowther D, (2005), A steady-state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements, *Building Services Engineering Research and Technology*, 26(4): 301-314.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (submitted), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air*.

Vihna J, Mikkil A, Valovirta I, Lindberg R, (2002), *Water vapour resistance of materials as a function of humidity*, Building Physics 2002- 6th Nordic Symposium, http://www.ivt.ntnu.no/bat/bm/buildphys/proceedings.php3, accessed: Oct 2006.

The Engineering Tool Box, *Thermal conductivity of some common materials,* www.engineeringtoolbox.com/thermal-conductivity-d_429.html, accessed: Jan 2007.

CHAPTER 6:

COMPARISON OF FIELDWORK RESULTS WITH BED PREDICTIONS

CHAPTER 6: COMPARISON OF FIELDWORK RESULTS WITH BED PREDICTIONS

6.1 Introduction

This chapter discusses the comparison between fieldwork data and the predictions of the BED model (Pretlove *et al.*, 2005). BED is a steady-state one-dimensional hygrothermal model, which predicts the average monthly temperature and RH within the bed core (the occupied space between mattress and covering), given the average monthly temperature and RH of the bedroom. Monitored values can be used for the bedroom conditions, if available. Alternatively, values predicted by models such as Condensation Targeter II could be used (Oreszczyn T and Pretlove S, 1999). This chapter only aims to assess the validity of the BED model, since the validation of Condensation Targeter II has been discussed elsewhere (Oreszczyn T and Pretlove S, 1999), and it exceeds the objectives of this thesis. Chapter 3 provides further details of the BED model.

Pretlove *et a.l* (2005) already carried out a preliminary validation exercise of the BED model, by comparing its predictions with the average monthly hygrothermal conditions measured in 3 beds (bed cores) over a year. The authors examined the predictions that the BED model provided when using measured bedroom data (Fig 6.1.1-2), as well as the predictions that the model provided when using bedroom conditions as predicted by Condensation Targeter II.

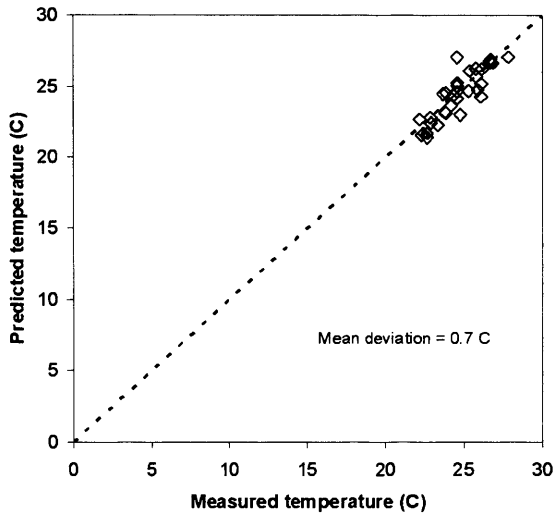


Fig. 6.1.1 Pretlove *et al.*, 2005: Comparison between monitored and modelled temperature in 3 beds over 1 year, using actual bedroom hygrothermal conditions as inputs.

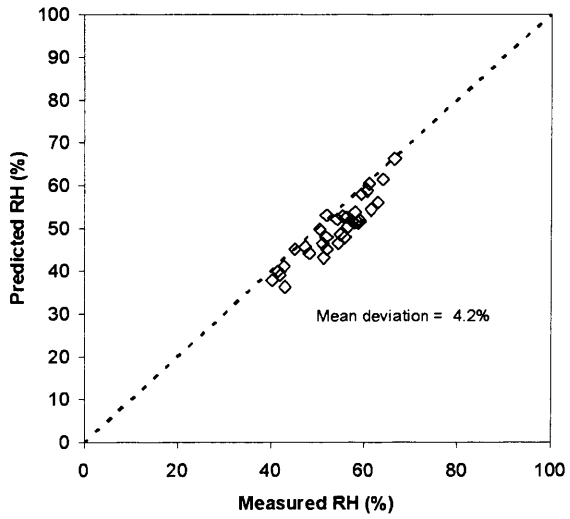


Fig. 6.1.2 Pretlove *et al.*, 2005: Comparison between monitored and modelled RH in 3 beds over 1 year, using actual bedroom hygrothermal conditions as inputs.

Figure 6.1.1 and 6.1.2 (from Pretlove *et al.*, 2005) show that the root mean square error for the temperatures is 0.7 ^{0}C, and for the RH is 4.2%. These values show a reasonable agreement with the field data. However, Figure 6.1.2 also shows that there is a consistent tendency for the model to slightly under-predict relative humidity. Pretlove *et al.* (2005) conclude that: "*there are a number of reasons why this might be happening, including assumptions relating to material properties of the bedding, comfort, sweating, and heat and moisture transfer*".

It should be noted that in Figure 6.1.1 and 6.1.2 thirty-six data-points are presented, corresponding to 12 months for 3 beds. However, these data-points are not all independent from each other, since assumptions made for one bed will affect 12 data-points. It is therefore desirable to compare the BED predictions with a greater number of independent data-points (in this case, 12 different beds), as illustrated in the following sections.

6.2 Methodology and assumptions

The fieldwork data utilised in this section for the assessment of the BED's predictions are the average temperatures and RHs measured in the "bed cores" (mattress top surface, area under the chest) of the Series 1 and the Series 2 beds[1]. Series 3 data is not included for 2 main reasons: a) Series 3 monitored children, who might be governed by different thermo-regulatory processes than adults, whilst the BED model is intended for adults; b) an additional padded cover was used to cover the bed sensors, which represents a confounding element when analysing the monitored bed results.

As already illustrated in Chapter 4, Series 1 included monitoring of 9 bedrooms and beds for 6 weeks, with the bed sensor placed on the top bed surface, under the chest area ("bed core"). However, data from one of the Series 1 beds had to be ignored, since the bed sensors broke. The Series 2 study included monitoring 4 bedrooms and beds for 6 weeks, with a greater number of sensors located in the bed, including the "bed core" location. So, 12 independent data-points (bed core measurements) were available overall, for comparison with the BED predictions.

[1] See Chapter 4 for a description of fieldwork protocol.

It must be also mentioned that in both Series 1 and Series 2, the sensors were placed in direct contact with the mattress, and were then covered by a mite-proof cover (standardised for Series 1 and 2), as well as by bedding sheets (not standardised). The duvets (and the pillows) were not standardised. The same mattress type and make was used for the Series 2 study.

The BED model requires a number of input variables, described in more details in Chapter 3 and by Pretlove *et al.* (2005). Quite a few of these variables could not be measured in the fieldwork study, due to practical constraints. Pretlove *et al.* could not measure many of those input variables either and they used published values, for example for the properties of the mattress and of the cover. However, a sensitivity analysis carried out by Pretlove *et al.* (2005, Fig 6.2.1) suggested that the RH in the bed core is mostly dependant on parameters affecting room conditions, rather than parameters specific to the BED model. Consequently, the uncertainty in some input variables for the BED model may not result in large discrepancies between predicted and measured values. Nonetheless, the impact of such uncertainties was estimated by adopting the Differential Sensitivity Analysis (DSA) method (Lomas and Eppel, 1992), which was reviewed in Chapter 2.

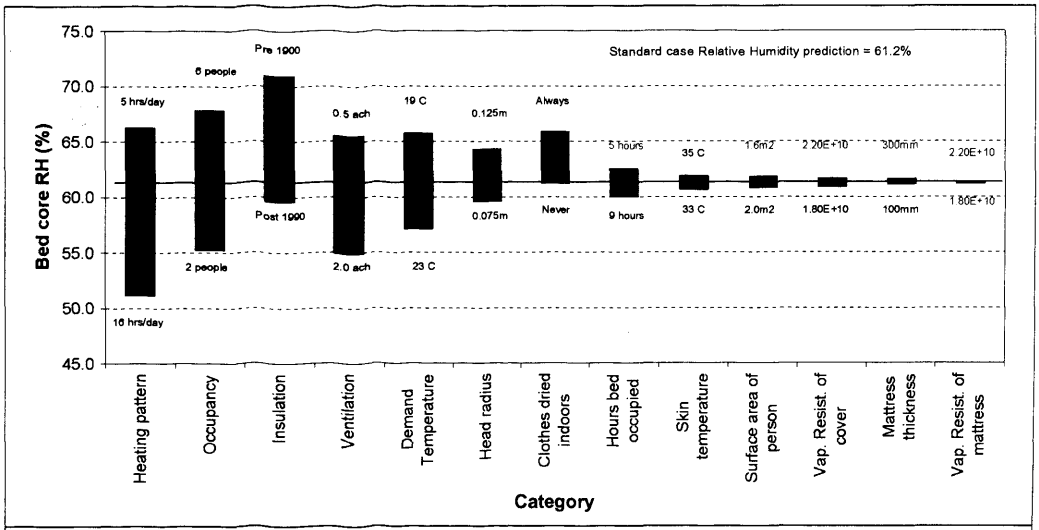


Fig 6.2.1 Sensitivity analysis of the BED model (Pretlove *et al.*, 2005)

In order to simulate fieldwork data, each input variable was assigned a base-case value, which corresponded to the measured results – when available, for example conditions in the bedroom – or to the same correspondent value utilised by Pretlove *et al.* (2005) in their analysis. Although mattress type and thickness had

been recorded in the fieldwork study, the hygrothermal properties of each mattress could not be measured. However, most mattress in the field study were sprung type. Therefore, the hygrothermal properties of the mattress simulated in BED were assumed to be the same as the Series 2 (sprung) mattress. The most relevant properties - except the heat capacity - of the materials constituting the Series 2 mattress had been measured by the Centre for Technical Textiles, University of Leeds. As for the properties of the air cavity in the sprung mattress, these had to be derived from available published data - as already discussed in Chapter 5.

Since the mattress in the BED model is homogenous, the overall thermal conductivity and vapour resistivity of the Series 2 mattress were calculated, where:

$$k_{mattress}=d_{mattress}*(\Sigma TR_i)^{-1} \quad [6.2.2]$$

$$r_{mattress}=(\Sigma VR_i)^{*}(d_{mattress})^{-1} \quad [6.2.3]$$

where

- $k_{mattress}$ is the mattress thermal conductivity ($Wm^{-1}K^{-1}$);
- $d_{mattress}$ is the mattress total thickness (m);
- TR is the thermal resistance of each layer (m^2K/W);
- $r_{mattress}$ is the mattress water vapour resistivity ($Nskg^{-1}m^{-1}$);
- VR is the water vapour resistance of each layer (m^2sPa/kg).

It should be mentioned that the thermal surface resistance was not included in this calculations, since this is taken into account separately in the model. The surface vapour resistance is assumed as negligible in the BED model.

Table 6.2.1 shows the main inputs used in the BED model, indicating which variable was measured (or estimated based on measurements). The variable "number of hours in bed per day" was not *measured* as such, but participants were interviewed and asked how many hours they slept on average per day.

Table 6.2.1 Input parameter for the base case and correspondent input changes Δi for the Differential Sensitivity Analysis.

	Base case	Input Change Δi (2.33xst. dev.)
BASIC DATA:		
Bed occupant metabolic rate (Wm^{-2})	40	10
Surface area of occupant (m^2)	1.8	0.2
BED OCCUPANT GAINS:		
Number of hours in bed per day (h)	Measured*	1
MATTRESS PROPERTIES:		
Thermal conductivity ($Wm^{-1}K^{-1}$)	0.10#	0.01
Vapour resistivity ($Nskg^{-1}m^{-1}$)	3.94E+09#	2.84E+09
Thickness (m)	Measured	0.01
COVER PROPERTIES:		
Thermal conductivity ($Wm^{-1}K^{-1}$)	0.04	0.02
Vapour resistivity ($Nskg^{-1}m^{-1}$)	2.0E+10	2.0E+09
OCCUPANT PROPERTIES:		
Skin surface temperature (°C)	34	1
Body vapour resistance ($Nskg^{-1}$)	7.9E+08	1.58E+08
SURFACE HEAT RESISTANCES:		
Cover (m^2KW^{-1})	0.1	0.02
Mattress (m^2KW^{-1})	0.17	0.03
BEDROOM MONTHLY CONDITIONS:		
Average bedroom temperature (°C)	Measured	0.2
Average bedroom relative humidity (%)	Measured	3

* Average number of hours in bed per day, as reported by the fieldwork participant in the interview. #Partly measured and party estimated, see Chapter 5.

Table 6.2.1 also lists the input changes Δi utilised for each input parameter for the Differential Sensitivity Analysis. These input changes were based on those assumed by Pretlove *et al.* (2005) in their sensitivity analysis (Fig 6.2.1), except for the parameters marked as "measured". For the measured parameters the Δi was estimated based on *ad hoc* considerations. For example, the Δi for the reported number of hours in bed was assumed to be 1 hour, since it was considered improbable that the volunteers would overestimate or underestimate this by more than this quantity. For the Δi of the temperature and RH inputs (measured bedroom conditions), the manufacturer's quoted accuracy was assumed to be equivalent to +/-2.33 x standard deviations, as already explained in Chapter 5. Finally, the Δi for the mattress properties was the one estimated in Chapter 5.

The next section illustrates the BED predictions, in comparison with the fieldwork data.

6.3 BED predictions and fieldwork data: results

In this section the BED predictions are compared with the monitored fieldwork results.

Figure 6.3.1 and 6.3.2 show the comparison between predicted and monitored results, including the error bars due to the uncertainties. In particular, the vertical bars correspond to errors in predictions due to uncertainties in input variables, whilst the horizontal bars correspond to error measurement in the results due to sensors inaccuracies. The graph shows that the root mean square error is 0.9 °C for the temperature, and 6.1 % for the relative humidity. Although these values are a little higher than those found by Pretlove *et al.* (2005) (Fig 6.1.1-2), these results are still reasonable when considering the uncertainties of the input variables. In fact, once these are taken into account, most temperature and RH data points fall within measured results. A slight tendency for over-predictions - particularly for the RH – can also be observed. This is the opposite of what Pretlove *et al.* (2005) found, as illustrated in Figure 6.1.2. The dust-proof cover positioned between the sensor and the body in the fieldwork mattresses for Series 1 and 2 might account for these over-predictions - at least in part. However, it should also be pointed out that in Figure 6.3.1 and 6.3.2 the uncertainties due to error measurements in the temperature and RH results (horizontal bars) are larger than the uncertainties due to input variables inaccuracies (vertical bars). Therefore, in order to assess the model's predictions more accurately in relation to the measurement results, it is recommended that dataloggers with a better accuracy are utilised in future studies. Ideally, the data illustrated in this chapter should have been plotted together with the data from Pretlove *et al.* (2005), which however was not available to the author.

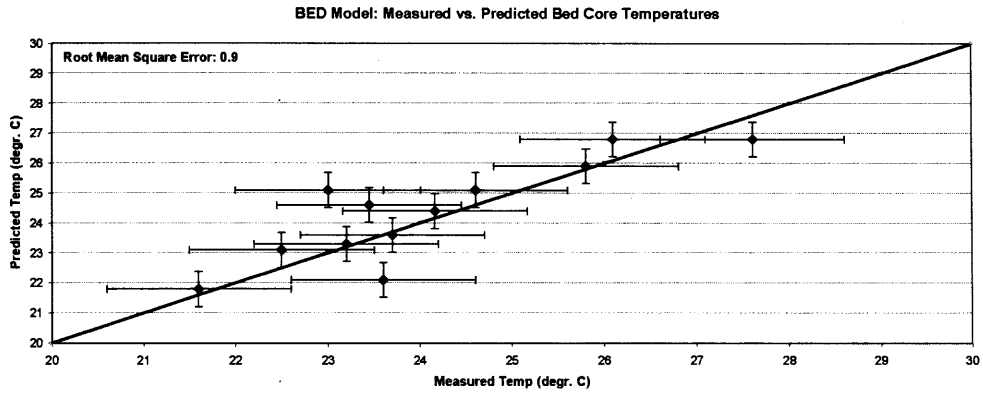


Fig. 6.3.1 Fieldwork data (Series 1 and 2), measured versus BED-predicted temperatures.

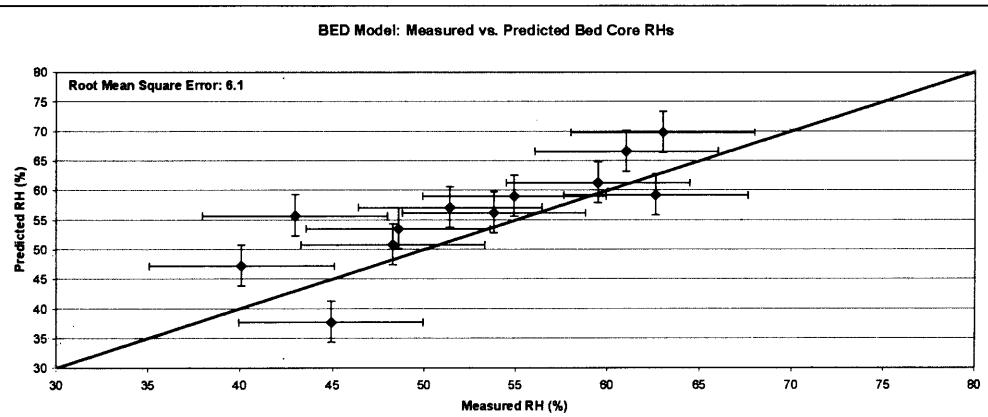


Fig. 6.3.2 Fieldwork data (Series 1 and 2), measured versus BED-predicted relative humidities.

The following section summarises the findings of this chapter.

[3] Software for the simultaneous heat and moisture transport in building components, Fraunhofer Institute for Building Physics.

6.4 Summary discussion

This chapter compared the predictions of the BED model with the results from twelve beds monitored for six weeks during the fieldwork study (eight Series 1 beds, and four Series 2 beds). The results showed that the root mean square error for the temperature data is 0.9 ^{0}C and 6.1 % for the RH data. These results are not significantly dissimilar from a similar exercise carried out by Pretlove *et al.* (2005), who monitored three beds over 1 year. However, Pretlove's study concluded that the model's RH predictions appeared consistently lower than the field data, while an opposite trend could be observed in this study. On the other hand, if the uncertainties associated with input variables and with error measurements are taken into account, most temperature and RH data points fall within the measured results. The uncertainties due to error measurements were larger than the uncertainties due to input variables inaccuracies. Therefore, in order to assess the model's predictions more accurately, it is recommended that dataloggers with a better accuracy are utilised in future studies.

Finally, it is worth mentioning that the BED model currently predicts bed conditions correspondent to the *surface* of the mattress, under the chest area. Pretlove *et al.* (2005) suggest that the bed core can be used as an overall indicator of colonisation risk for the mattress as a whole, where if average conditions at this point are favourable for mite population growth, then they are likely to be favourable elsewhere in the mattress. Pretlove *et al.* also conclude that if average conditions are clearly unfavourable at this point, then they are likely to be unfavourable elsewhere within the mattress. However, this is not necessarily true, since Pretlove *et al.* also point out that detailed measurements showed that other positions with the mattress may be more favourable for mite colonisation - provided physical access is achievable at these other locations and food is accessible. Therefore, it may be desirable to modify the BED model so that it can also predict hygrothermal conditions for the mattress zone which is more

[4] The Hobo H8 dataloggers used in the eight Series 1 beds had an accuracy of 0.7 ^{0}C for the temperature, and of 5% for the RH. The Type K thermocouples used for the four Series 2 beds had an accuracy of 1 ^{0}C, while the RH sensors (Honeywell HIH-3610 Series) had an accuracy of 2% (although the RH sensors had been calibrated, under steady state conditions).

favourable to mite growth (1-2 cm below the top mattress surface, see Ridley *et al.*, submitted). This can be easily achieved, once the bed core hygrothermal conditions and the mattress properties are known. The hygrothermal conditions within the depth of the mattress could be calculated by using the Glaser method (BSI, 2002), which takes into account the resistance of each mattress layer. However, since in the BED model the mattress is assumed as a homogeneous layer, simple interpolation could also be used. If the BED model predicted those 2 data points within the mattress, this would give an indication of best-case scenario (i.e. bed core) and worst-case scenario (i.e. 1-2 cm below the chest area) for mite population growth in the mattress. This issue is addressed further in Chapter 10.

Chapter 6: References

BSI, (2002), *BS EN ISO 13788:2002. Hygrothermal performance of building components and building elements. Internal surface temperature to avoid critical surface humidity and interstitial condensation. Calculation methods.* London: BSI.

Lomas and Eppel, (1992), Sensitivity analysis techniques for building thermal simulation programs, *Energy and Buildings*, 19: 21-44.

Oreszczyn T and Pretlove S E C, (1999), Condensation Targeter II: Modelling surface relative humidity to predict mould growth in dwellings, *Building Services Engineering Research and Technology,* 20 (3) 143 – 153.

Pretlove SEC, Oreszczyn T, Ridley I, Wilkinson T, Crowther D, (2005), A steady-state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements, *Building Services Engineering Research and Technology*, 26(4): 301-314.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (submitted), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air.*

CHAPTER 7:

COMPARISON OF FIELDWORK RESULTS WITH POPMITE PREDICTIONS

CHAPTER 7: COMPARISON OF FIELDWORK RESULTS WITH POPMITE PREDICTIONS

7.1 Introduction

This chapter discusses the comparison between fieldwork data (caged mites) and the predictions of the Popmite model (Biddulph *et al.*, 2007). Popmite is a model which predicts the effects of hygrothermal conditions on each life cycle phase of a DP house dust mite (i.e. eggs, juvenile, adults). The *Popmite Version 7d* is utilised in this thesis, which is a further development of the model described by Biddulph *et al.* (2007), for predicting the impact of *transient* hygrothermal conditions on *wild* mites (see Chapter 3). Firstly, *Version 7d* utilises experimental data on wild DP mites, reared on a diet of skin and dust (Hart *et al.*, 2007) - whilst the previous versions adopted published data on lab DP mites. Furthermore, in *Version 7d* mite survival is related to the temperature-dependent Critical Equilibrium Humidity (CEH). If RH is above CEH, mites take up moisture from the air, eat and lay eggs at a given rate. If RH is below CEH, mites gradually stop absorbing moisture, eating and laying eggs, eventually dying. Like in the previous version of Popmite, temperature affects mite development rate and fecundity. In *Version 7d* eating rate has also been introduced, which is dependent on hygrothermal conditions.

In this chapter, the mite bags results are presented (section 7.3), and compared with Popmite predictions (section 7.4). The methodology utilised in this chapter is illustrated in section 7.2. The chapter ends with a summary discussion (section 7.5). It may be useful to remind the reader that the 'mite bags' are a new technique, whereby live DP mites are encapsulated with surplus food in sealed 'mite bags', made from allergen and mite proof porous material. The mite bags (similar in size and shape to a tea-bag) are placed in the bed (or the bedroom) next to a sensor recording the hygrothermal conditions to which they are exposed. After approximately six weeks, the bags are removed and the remaining number of live mites found in each bag can be compared with the Popmite predictions.

The next section provides further information on the fieldwork data, as well as on the assumptions utilised for the comparison between predicted and measured results.

7.2 Methodology and assumptions

The methodology for the fieldwork study was illustrated in Chapter 4. However, in this section some details of the methodology are highlighted, which are most relevant to the topic of this chapter. The fieldwork study was divided into 3 successive 'series', where the hygrothermal conditions of a number of beds (and corresponding bedrooms) were monitored for approximately 6 weeks. In each mite bag location, 3 bags were installed, for repeatability purposes. In Series 1, the hygrothermal conditions of nine beds (and bedrooms) were monitored[1], with the mite bags and related loggers[2] located on the top surface of the mattress (under the sheets), in the area corresponding to the occupant's chest when the bed is occupied. In each Series 1 mite bags, 50 adult laboratory-reared DP mites (1:1 males and females) were encapsulated, together with food (1:1 by weight liver and yeast). In Series 2, four beds/bedrooms were monitored, with a greater number of mite bags and loggers[3] located in each bed (8 locations in each bed), and with a set of mite bags also located in the bedroom (next to the bedroom logger). In each mite bag utilised in Series 2, 20 adult *wild* DP mites (1:1 males and females) were encapsulated, together with "wild" food (1:1 by weight skin and dust). In Series 3, 12 beds/bedrooms were monitored, with a set of mite bags placed in the bedroom (and in the living room in 2 dwellings). In addition, a set of mite bags with loggers[2] was placed on the top surface of the mattress (under a padded cover), in the area where the occupant's feet are located when the bed is occupied. Furthermore, additional mite bags (and loggers) were positioned in five of the twelve Series 3 beds, under the pillow and under the chest area (top mattress surface, under padded cover). As in Series 2, in each mite bag utilised in Series 3, 20 adult *wild* DP mites (1:1 males and females) were encapsulated, together with "wild" food (1:1 by weight skin and dust).

Overall, 82 sets of mite bags were installed. However, due to some data loss (e.g. breakage of hygrothermal sensor), 77 sets could be considered for the comparison with the Popmite predictions.

[1] The sensor in one of the beds stopped working, therefore only data for 8 beds is available from Series 1.
[2] Hobos, H8 Series: www.onset.com.
[3] Type K thermocouple and Honeywell HIH-3610 Series, from RS Components.

In all three fieldwork Series, after each set of mite bags was in place for approximately six weeks the bags were retrieved and the remaining number of live mites found in each bag was counted. These results can be compared with the Popmite predictions. The input data required in Popmite for this purpose is the transient hygrothermal conditions to which the mites were exposed, and the initial number of mites in each bag. In addition, Popmite also requires species-specific information on the effect that hygrothermal conditions have on each life cycle phase of a house dust mite. As already mentioned, this information (e.g. egg development and fecundity) had been obtained by the model developer (Phillip Biddulph) from experimental data on individual wild DP mites feeding on skin and dust and held at a range of constant hygrothermal conditions (Hart *et al.*, 2007). However, other input data required for *Popmite Version 7d* (e.g. Critical Equilibrium Humidity, feeding rates, etc.) had been obtained from published values, or estimated on the basis of various sources.

In all three fieldwork Series, the mite bags were transported to and from locations in sealed containers with NaCl saturated salt solution, which produces an ideal environment for the mites of approximately 75% RH (varying slightly with changes in temperature). After the mite bags were produced, they were installed in beds after 1-2 days maximum. In Series 1 and 2, once the bags were removed from the bed and transported to the lab, the mites were kept at 10 °C in an incubator (which inhibits mite growth) and the remaining live mites were counted within 48 hrs. The exact hygrothermal conditions were only recorded whilst the bags were in the beds. Consequently, the input hygrothermal data utilised in Popmite does not include the transport to and from the lab, nor the 48 hours in the incubator. It was assumed that this would not significantly affect the results, since the gaps in hygrothermal data were rather short. However, in the Series 3 study, the mites stayed in the Series 3 bags for a little longer than in the other two Series (approx. 10 days more), since the counting of live mites for Series 3 was carried out by an acarologist in Scotland. Therefore, in Series 3 the hygrothermal conditions to which the mites were exposed *after* being removed from the bed were monitored until the mites were counted. These recordings were included in the hygrothermal input data utilised in Popmite for Series 3.

Before presenting the comparison between the Popmite predictions and the mite bags counts, the uncertainty in the measurements and in the predictions must be discussed. In the methodology utilised for this study, there are a number of sources of uncertainty, some of which can be estimated more easily than others:

1) The hygrothermal measurements utilised as inputs in Popmite are subjected to uncertainties due to sensor accuracy, which would affect the Popmite predictions.

2) Some uncertainty is associated with the actual number of live mites present in the mite bags at the beginning and at the end of the monitoring period. This is due to a number of reasons. Firstly, in each monitored location 3 mite bag replicates were installed. At the end of the 6 weeks period, the number of live mites counted in each bag varied slightly. This variation is likely to be due to natural biological variations, to the health status and the age of the adult mites which were originally sealed in the bags. Therefore, when comparing the Popmite predictions with the live mites counts (average of 3 mite bags), the standard deviation from the 3 bags should also be taken into account. Secondly, any potential inaccuracies in mite counting are difficult to estimate, and might at least in part account for the variability in mite counts for those bags placed in the same location. Thirdly, although great care was taken when the adult mites were counted and sealed in each bag[4], it cannot be excluded that a couple of eggs or juvenile mites may have occasionally be sealed in the bag too. Again, this effect is difficult to estimate, but it may in part be accounted for by the 3 replicate bags.

3) Some species-specific information is required in Popmite, which is not available in the published literature. Consequently, this information had to be estimated by the model developer, based on several sources. These uncertainties are also likely to have an impact on the Popmite predictions. This issue is addressed further in the sensitivity analysis for Popmite (Chapter 9).

4) Popmite requires the input population age structure to be specified (i.e. how far into its development each mite is). However, it is difficult to determine this

[4] Mr Toby Wilkinson (Cambridge University) produced the mite bags.

information, and it was assumed that the population in the mite bags had a "spread of all ages". Nonetheless, if the sample was taken from a young population, this assumption might be incorrect. The impact of this issue is discussed later in this chapter.

Most of the above uncertainties is rather difficult to estimate. When testing the validity of the hygrothermal bed models, the Differential Sensitivity Analysis (DSA) method was utilised, in order to assess the impact of uncertainties on the model's predictions (see Chapter 5). However, it was considered inappropriate to adopt this method for Popmite, since the DSA requires a certain degree of knowledge of likely distribution of input parameters. However, insufficient information is available for some crucial input parameters in Popmite – particularly those associated with eating rates. Therefore, in this chapter only the effect of error measurement in hygrothermal conditions is considered, together with the variability in mite counts for the replicates placed in the same locations. This variability is probably due to biological variability but might also reflect possible inaccuracies in mite counts.

The next section illustrates the mite bag results.

7.3 Mite bags results

In this section the mite bags results are illustrated, in relation to the hygrothermal conditions to which they were exposed. As previously mentioned, in each mite bag location 3 mite bag replicates were installed (mite bag set). Laboratory studies have shown that laboratory-reared mites have stronger reproduction and development than wild mites, except when under environmental stress, and that diet is a significant factor, particularly in sub-optimal conditions (Hart *et al.*, 2007). Since the Series 1 mite bags contained 50 laboratory-reared mites on a laboratory diet- as opposed to 20 wild mites on a "natural diet" as in the Series 2 and Series 3 - the results from the eight Series 1 mite bags sets are excluded from the graphs[6]. For each of the remaining 69 mite bag sets (from Series 2 and Series 3, beds or bedrooms), the results are here presented in terms of average (of the 3

[5] Apart from the properties of the air gap of the sprung mattress

[6] The sample size of Series 1 mite bags was to small for detecting a noticeable difference between Series 1, and Series 2 and 3 results.

mite bags) total live mites (i.e. juveniles plus adults)[7]. For each mite bags set, the hygrothermal conditions presented in the graphs correspond to the average conditions to which the mite bags were exposed over the 6 weeks monitoring period. The percentage of time the measured RH was above the Critical Equilibrium Humidity (CEH) was also calculated ('% time RH> CEH'). It should be mentioned that the CEH for DF mites was found to be temperature-dependent (Arlian and Veselica, 1981). No published information is available on the CEH of wild DP mites. However, in Popmite 7d the CEH for wild DP mites was calculated based on relevant experimental data (Hart *et al.*, 2007).

Figure 7.3.1 shows the correlation between the average number of mites found in the mite bags after 6 weeks, and the correspondent: 1) average RH; 2) '% time RH> CEH', based on published CEH information for DF mites[8] (Arlian and Veselica, 1981); 3) '% time RH> CEH', based on the Popmite CEH[9] values for DP wild mites. The red line in Figure 7.3.1 corresponds to the starting population in the mite bags (20 mites). Therefore, for any data point above the red line, growth occurred in the mite bags population after 6 weeks. Figure 7.3.1 shows that the % of time above CEH is a slightly better predictor (in terms of R-squared value) of measured mites, than the average measured RH. The graph also shows that growth in the mite bags only occurred at average RHs of approximately 55% or greater. This finding is in line with some published information (Arlian *et al.*, 2001). However, it should also be pointed out that the data illustrated in the graph is affected by temperatures as well. For example, some mite bags were exposed to hygrothermal conditions where the RH was always above CEH, but since the temperature was rather low (15-16 °C), the final mite count was also low (low temperatures increase development times). The data also show that the mite bags are an effective method, since under favourable conditions the caged population can grow.

[7] Eggs could not be counted in the mite bags.
[8] $CEH=41.7*e^{(0.014*temp)}$
[9] $CEH=54.5-0.005*temp+[525.6/(temp-39.3)^2]$, only valid if temp. < 37 °C

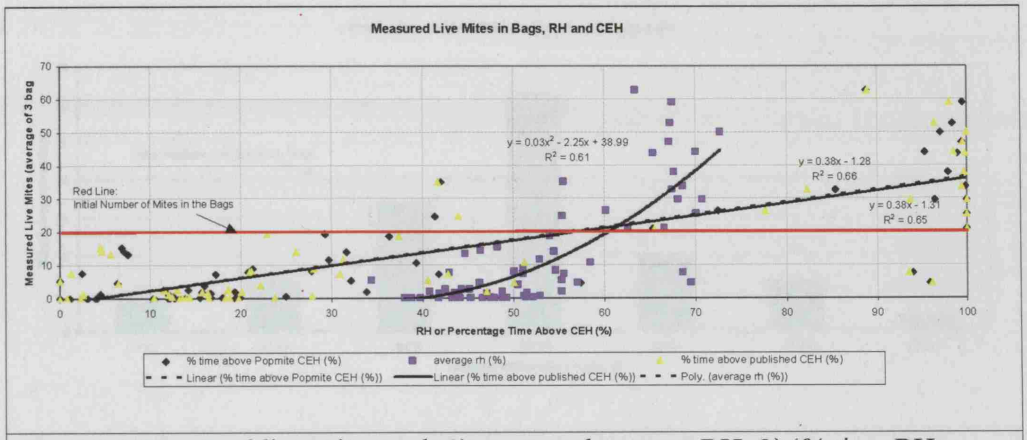


Fig 7.3.1 Measured live mites and: 1) measured average RH; 2) '% time RH > Popmite CEH; 3) % time RH > published CEH (Arlian, 1981).

Figure 7.3.2 shows the correlation between average total live mites, and measured average temperature[10]. As in the previous graph, the red line indicates the point above which growth occurred in the mite bags (after 6 weeks).

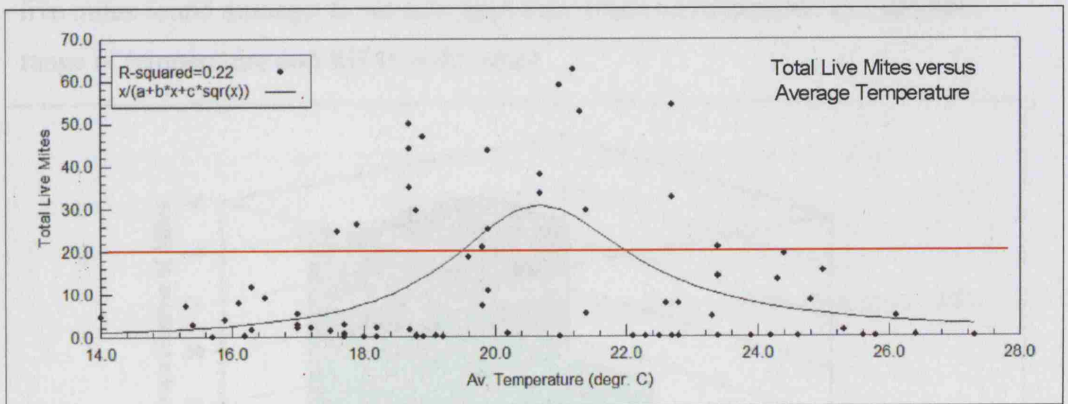


Fig 7.3.2 Average total live mites in mite bags and average temperature.

The graph indicates a tendency for the mite population to thrive better in mid-range temperature values (20-22 °C). This is illustrated further in Figure 7.3.3, which is a histogram showing the average of the mite bag results for a number of temperature intervals.

[10] The data fitting was carried out with the DataFit software (www.oakdaleengr.com/).

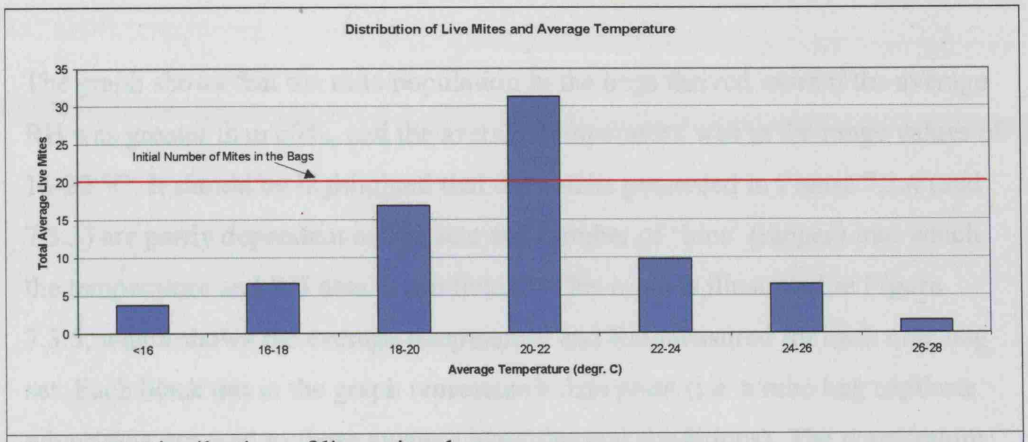


Fig 7.3.3 Distribution of live mites by average temperature.

The graphs presented so far only show one hygrothermal variable at a time. However, as previously mentioned both temperature and RH should be considered when assessing the most favourable conditions for dust mites[11]. Figure 7.3.4 shows the distribution of the mite bags results for the correspondent monitored temperature and RH values. Each block (z-axis) represents the average number of live mites found amongst those mite bags sets which were exposed to a specific range of temperature and RH (x and y axis).

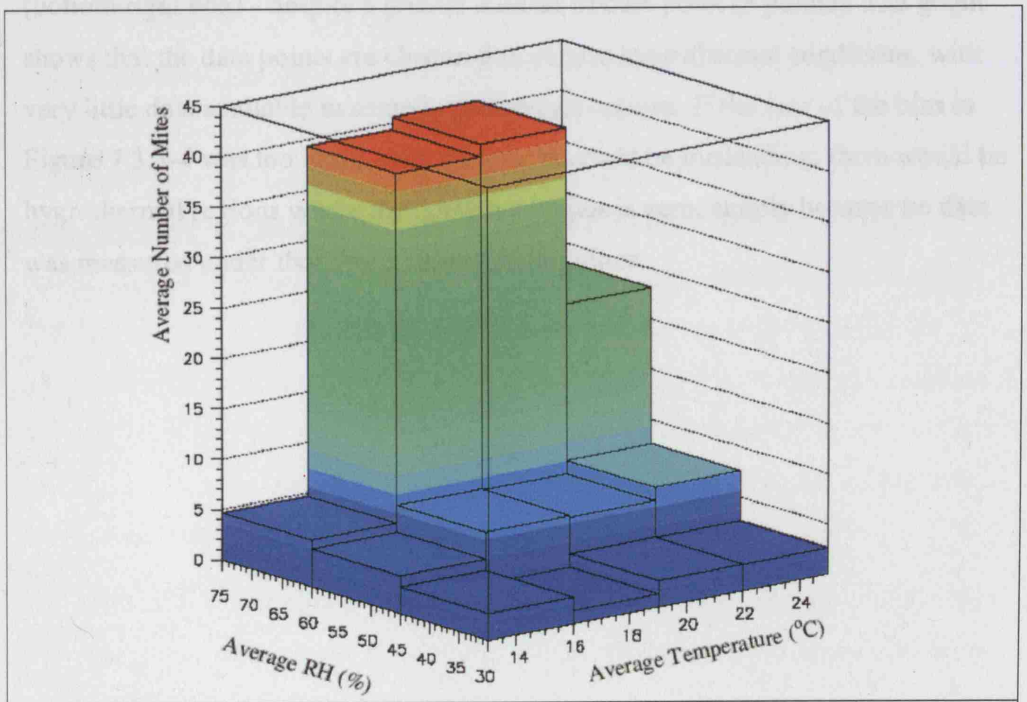


Fig 7.3.4 Distribution of total live mites measured in the mite bags after 6 weeks, versus average measured temperature and RH.

[11] If for example the RH is very low, then even favourable temperatures would not result in mite growth.

The graph shows that the mite population in the bags thrived more if the average RH was greater than 60%, and the average temperature was in the range values of 16-22 °C. It should be highlighted that the results presented in Figure 7.3.4 (and 7.3.3) are partly dependent on the size and number of 'bins' (ranges) into which the temperature and RH data is subdivided. This issue is illustrated in Figure 7.3.5, which shows the average temperature and RH measured for each mite bag set. Each black dot in the graph represents a data point (i.e. a mite bag triplicate which was exposed to those average hygrothermal conditions). The temperature and RH data is subdivided in the graph into a number of 'bins', which create a grid represented by blue lines. For each box of the grid, a red square is plotted, whose size schematically represents the average number of mites found in each temperature and RH 'bin'. For example, for RHs between 60-75% and temperatures between 22 and 25 °C (top right box), 5 data points are found, but the average number of mites (size of the red square) is bigger, for example, than in the case where the temperature is the same but the RH is between 30-45 % (bottom right box) - despite a greater number of data point (8 points). The graph shows that the data points are clustered in certain hygrothermal conditions, with very little data available in some hygrothermal regions. If the size of the bins in Figure 7.3.3-4 was too small, then the graphs could be misleading: there would be hygrothermal regions where the number of mites is zero, simply because no data was measured under those hygrothermal conditions.

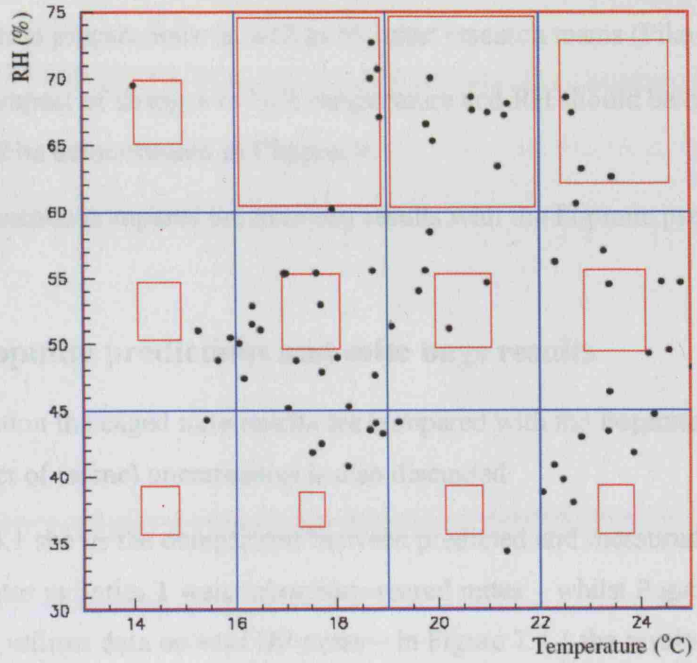


Fig 7.3.5 Hygrothermal distribution of mite bag results (see text for an explanation of the graph).

This section presented the mite bags results, in relation to the hygrothermal conditions to which they were exposed. The results confirm that the mite bags are an effective method, since population growth was observed under favourable hygrothermal conditions. The results also suggest that the mites in the bags thrived more if the average RH was greater than 55-60%, and the average temperature was 16-22 °C. It should be emphasised that the results illustrated in this section should be taken with some caution, in relation to the issue of the psychrometric control of house dust mites in dwellings. For example, it should not be concluded that the average temperature in the bedroom should be kept outside the 20-22 °C range (Fig 7.3.3), in order to avoid mite *growth* in beds. This is for three main reasons:

1. The mite bags results presented so far correspond to monitored conditions in beds (and some in rooms). Average conditions in a room do not necessarily correspond to average conditions in a bed.
2. Transient conditions - as opposed to average values – are important, particularly in certain circumstances (e.g. RH close to CEH). The importance of transient conditions will be highlighted in Chapter 10, and it has been

confirmed by laboratory studies carried out for the EPSRC-funded study (paper in preparation), as well as by other research teams (Pike *et al.*, 2005).

3. The impact of changes in *both* temperature and RH should be considered, as it will be demonstrated in Chapter 9.

The next section compares the mite bag results with the Popmite predictions.

7.4 Popmite predictions and mite bags results

In this section the caged mite results are compared with the Popmite predictions. The impact of (some) uncertainties is also discussed.

Figure 7.4.1 shows the comparison between predicted and measured results. Since the DP mites in Series 1 were laboratory-reared mites – whilst Popmite Version 7d mostly utilises data on *wild* DP mites – in Figure 7.4.1 the results from Series 1 are separated from the other two Series.

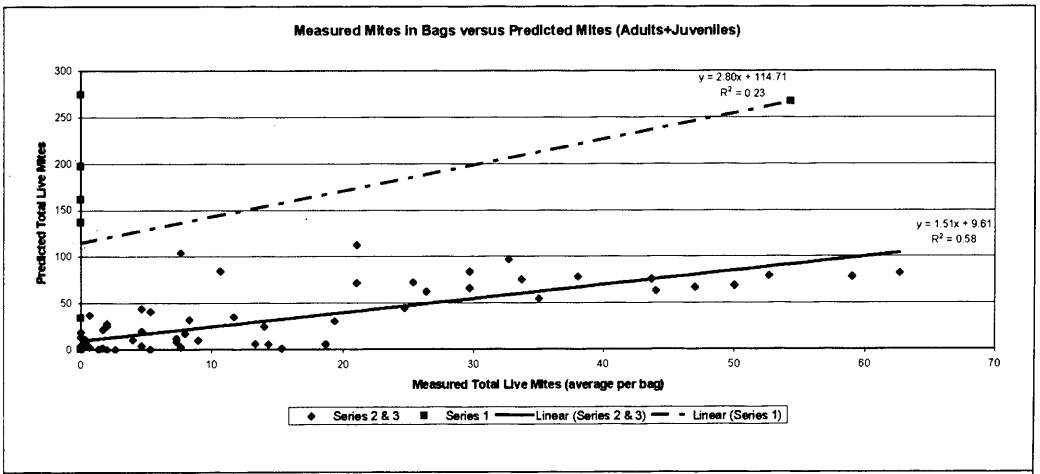


Fig 7.4.1 Measured versus predicted total live mites.

The results for Series 2 and 3 (wild mites) show that there is a moderate correlation between the measured and the predicted results (R^2=0.58), and that Popmite tends to over-predict by a factor of approximately 1.5. The R-squared value of 0.58 is rather promising, considering that Popmite is predicting biological phenomena, which usually have noticeable variability. The results for Series 1 are

[12] Software for the simultaneous heat and moisture transport in building components, Fraunhofer Institute for Building Physics.

less promising, which is unsurprising due to the differences between laboratory-reared and wild mites (Hart *et al.*, 2007).

In Figure 7.4.2 the comparison between measured and predicted Series 2 results is illustrated with the error bars due to error measurement (logger accuracy) and to the variability between the 3 mite bags in each location.

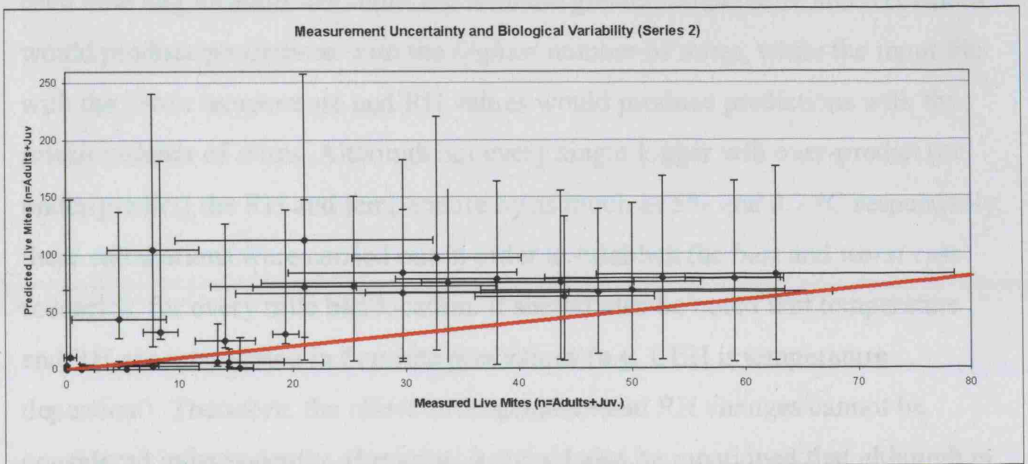


Fig 7.4.2 Measured versus predicted total live mites, in relation to measurement uncertainty (vertical error bars: impact of the maximum and minimum readings due to loggers inaccuracy) and to biological variability (horizontal error bars: standard deviation of the counts for the 3 bags in each location). The red line represent the ideal perfect fit.

As already mentioned in the previous section, in each monitored location 3 mite bag replicates were installed. At the end of the 6 weeks period, the number of live mites left in each bag varied slightly. In Figure 7.4.2 the horizontal error bars correspond to the standard deviation of the live mite counts from the 3 replicate mite bags in each location. This takes into account biological variability, and may also take partly into account errors in counting the mites, both when the bags were made and/or when the final counting was performed.

The vertical error bars in Figure 7.4.2 correspond to the uncertainty due to the logger accuracy. For example, the Hobo H8 series has an accuracy of +/- 5% RH over the range of 5 to 50 °C, and of +/- 0.7 °C at 21 °C[13]. In order to estimate the potential impact of these uncertainties on the model's predictions, for each hygrothermal input file two additional files were created. One of these files was

[13] These values vary little with changes in hygrothermal conditions, for the range considered in this study.

obtained by *adding* to every reading of the original input file the temperature and RH accuracy band (e.g. for RH=5%). The other file was obtained by *subtracting* to every reading of the original input file the temperature and RH accuracy band. The two additional files were then separately used as inputs in Popmite, producing a *band* of mite predictions for each monitored location. It was anticipated that for each mite bag location, the input file with the greater temperature and RH values would produce predictions with the *highest* number of mites, while the input file with the lower temperature and RH values would produce predictions with the *lowest* number of mites. Although not every single logger will over-predict (or under-predict) the RH and temperature by as much as 5% and 0.7 °C respectively, these calculations were carried out in order to establish the *best* and *worst* case scenarios, for every mite bag location. It should also be noted that temperature and RH are interrelated in Popmite predictions (e.g. CEH is temperature dependent). Therefore, the effect of temperature and RH changes cannot be considered independently. However, it should also be mentioned that although in theory higher RHs and temperatures should lead to greater mite numbers than low values, in some cases the Popmite predictions contradicted this principle. This is due to the interaction of a number of factors: the combined effect of temperature and RH on mite development times and feeding rates, as well as the temperature-dependency of the Critical Equilibrium Humidity.

Figure 7.4.2 shows that both the horizontal and vertical error bars can be rather large, particularly in some cases. If the hygrothermal conditions experienced by a mite bag are very close to CEH, even small changes in RH or temperature can lead to significant changes in final mite numbers. It could be argued that the way the effect of error measurement in hygrothermal conditions was calculated (worst and best case scenarios) leads to an overestimation of its impact. However, despite most error bars being rather large, in some cases these do not cross the ideal perfect fit line (in red in Figure 7.4.2). In most cases, the predictions are above the perfect fit line, confirming that Popmite is over-predicting.

Figure 7.4.3 compares the measured and the predicted results (for Series 2 and 3), distinguishing between adults and juveniles. Figure 7.4.3 shows that there is very little correlation between measurements and predictions for adult mites, while a good correlation exists for juveniles, although with over-predictions.

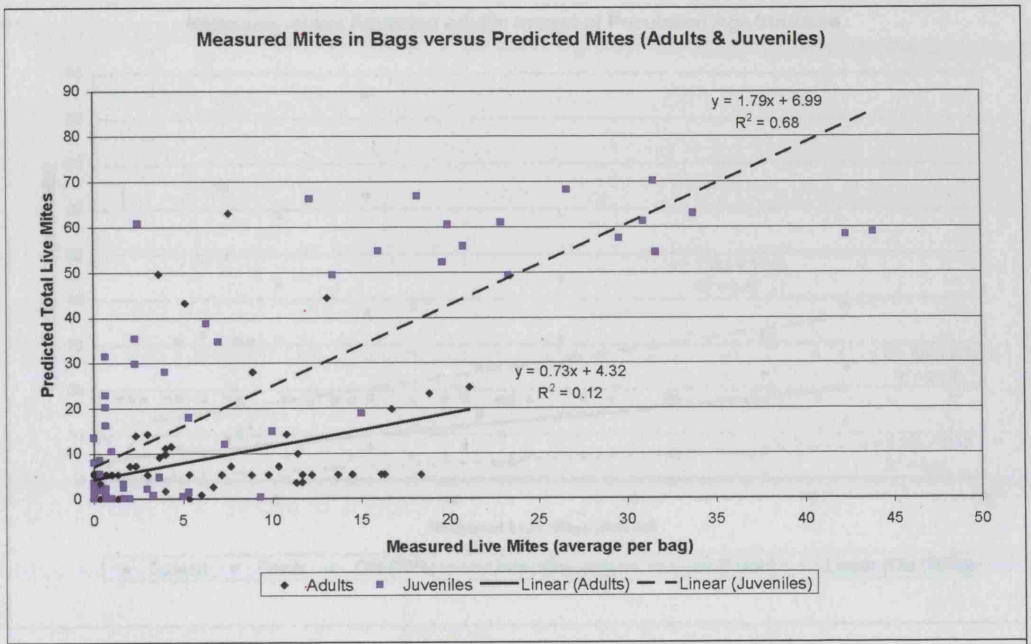


Fig 7.4.3 Series 2 and 3 (wild mites): comparison between measured and predicted results, juveniles and adults.

Another source of uncertainty for the comparison between predicted and measured results is the life cycle stage and the age of the mites in the mite bags. For the predictions in Figure 7.4.1 and 7.4.2 it was assumed that the initial mites were all adults, with an even spread of all ages. Figure 7.4.4 compares the predictions based on the assumptions of a spread of ages in the starting population, with the predictions based on the assumption that the mites in the bags were all "fresh" (i.e. just turned into adults), or were all "old" (i.e. 70% into their adult development).

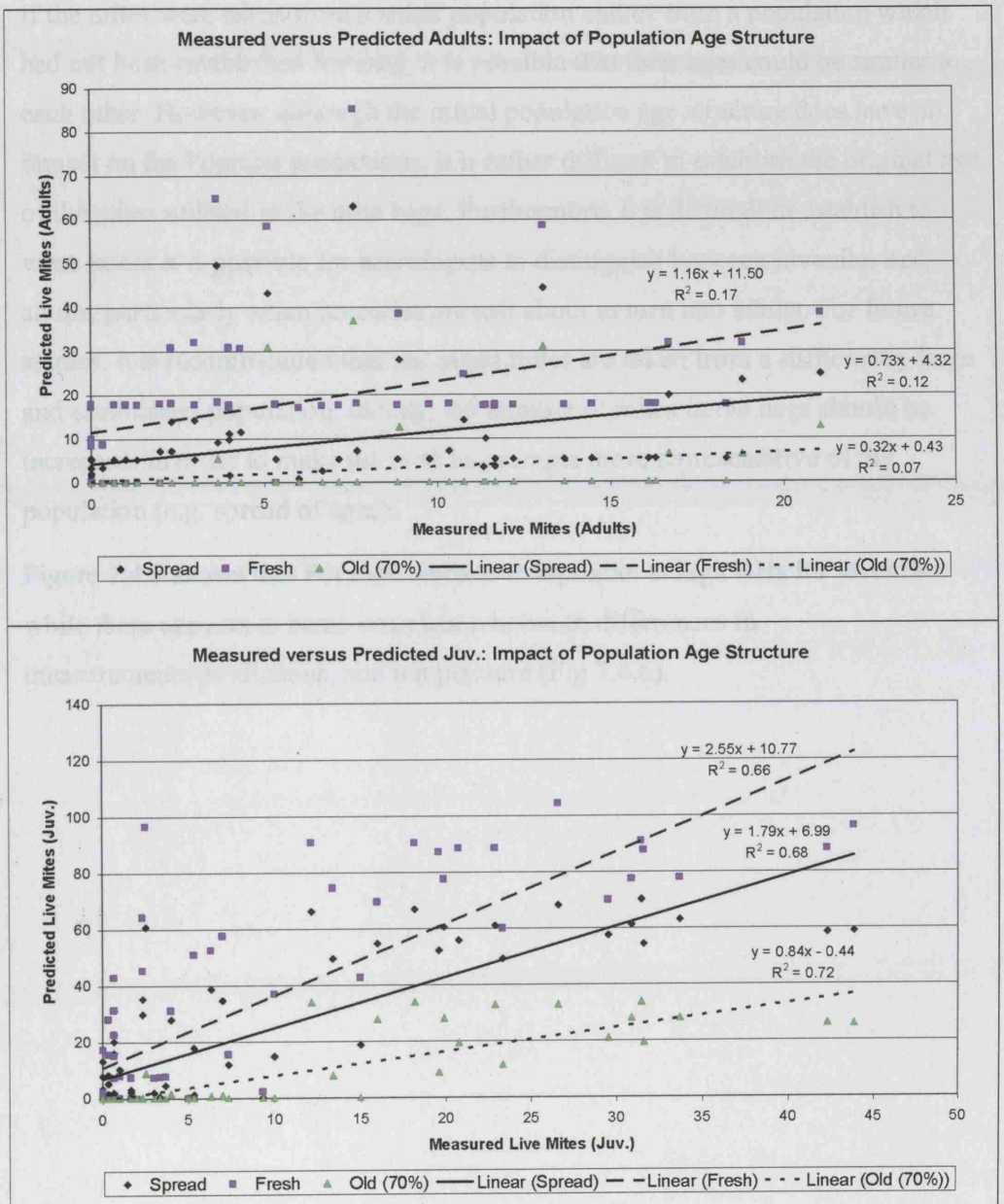


Fig 7.4.4a and 7.4.4b Impact of initial population age structure on comparison between measured and predicted mite counts.

Figure 7.4.4 shows that if it is assumed that the mites in the bags had *just* developed into adults (fresh mites), Popmite predictions would be even higher than when an even spread of ages is assumed. On the other hand, if the assumption was made that all mites are "old" (70% into their adult life), the

predictions are lower[14] and for the juveniles they are closer to the measurements. If the mites were taken from a small population and/or from a population which had not been established for long, it is possible that their ages could be similar to each other. However, although the initial population age structure does have an impact on the Popmite predictions, it is rather difficult to establish the original age of the mites utilised in the mite bags. Furthermore, it is difficult to establish to what extent it is possible for acarologists to distinguish between juveniles and adults, particularly when juveniles are just about to turn into adults. For future studies, it is recommended that the caged mites are taken from a sufficiently large and established population. Ideally, the number of mites in the bags should be increased, in order to make the mite bag sample more representative of the population (e.g. spread of ages).

Figure 7.4.5 shows that Popmite tends to overpredict at high RHs for juveniles, while there appears to be no correlation between differences in measurements/predictions, and temperature (Fig 7.4.6).

[14] This is expected, since it is logical that older mites would die faster and produce less offspring.

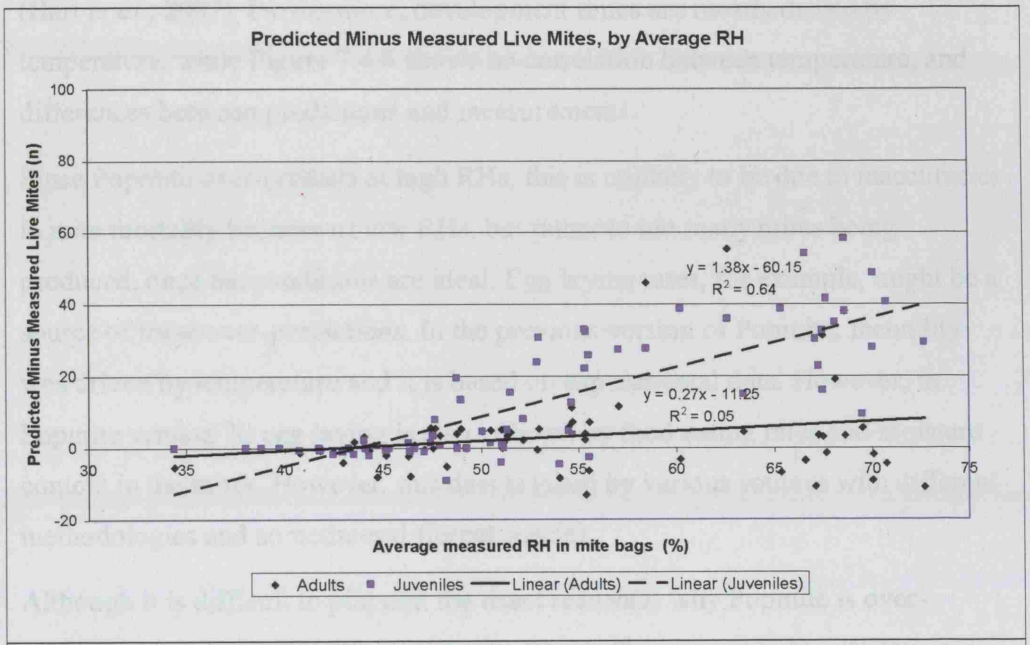


Fig 7.4.5 Difference between predicted and measured mite results, in relation to measured average RH.

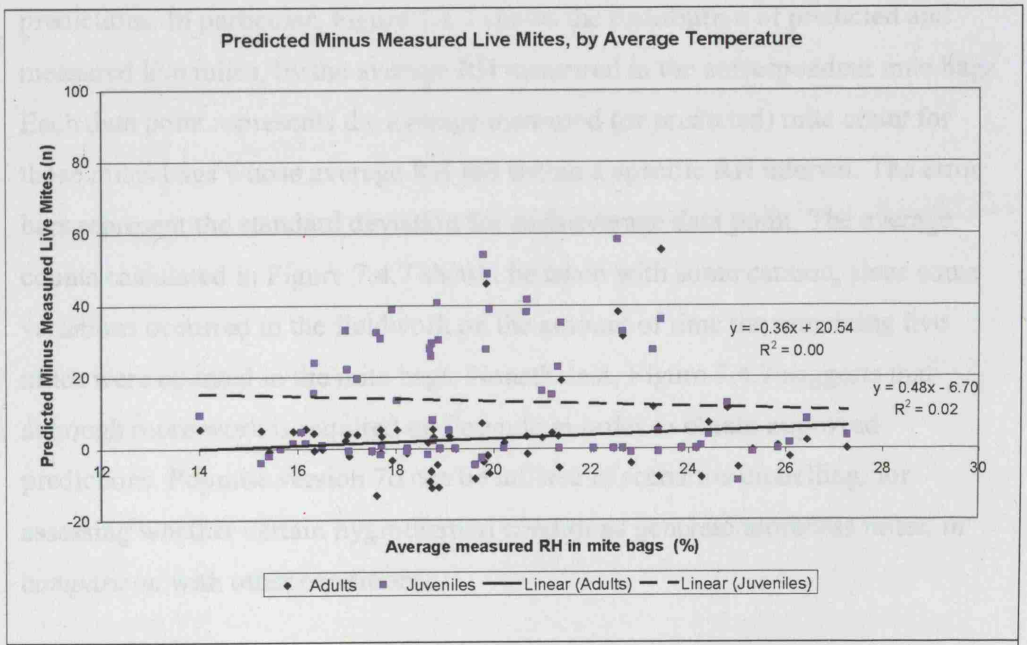


Fig 7.4.6 Difference between predicted and measured mite results, in relation to measured average temperature.

The Popmite over-predictions might be due to several factors. For example, development times (e.g. juveniles into adults) utilised in Popmite might be

incorrect. However, this data derives from experimental evidence on wild mites (Hart *et al.*, 2007). Furthermore, development times are mostly driven by temperature, while Figure 7.4.6 shows no correlation between temperature, and differences between predictions and measurements.

Since Popmite over-predicts at high RHs, this is unlikely to be due to inaccuracies in mite mortality because of low RHs, but rather to too many mites being produced, once the conditions are ideal. Egg laying rates, for example, might be a source of these over-predictions. In the previous version of Popmite, fecundity was driven by temperature and it is based on experimental data. However, in Popmite version 7d egg laying is also affected by food eating rates and moisture content in the mites. However, this data is taken by various sources with different methodologies and sometimes different species.

Although it is difficult to pinpoint the exact reason(s) why Popmite is over-predicting, a sensitivity analysis (Chapter 9) may draw some light onto this issue. However, Figure 7.4.7 shows that Popmite predictions tend to follow the same "direction" as the measurements, although the numbers are higher in the predictions. In particular, Figure 7.4.7 shows the distribution of predicted and measured live mites, by the average RH measured in the correspondent mite bags. Each data point represents the average measured (or predicted) mite count for those mites bags whose average RH fell within a specific RH interval. The error bars represent the standard deviation for each average data point. The average counts calculated in Figure 7.4.7 should be taken with some caution, since some variations occurred in the fieldwork on the amount of time the remaining live mites were counted in the mite bags. Nonetheless, Figure 7.4.7 suggests that although more work is required on Popmite in order to obtain improved predictions, Popmite version 7d can be utilised in scenarios modelling, for assessing whether certain hygrothermal conditions generate more/less mites, *in comparison* with other conditions.

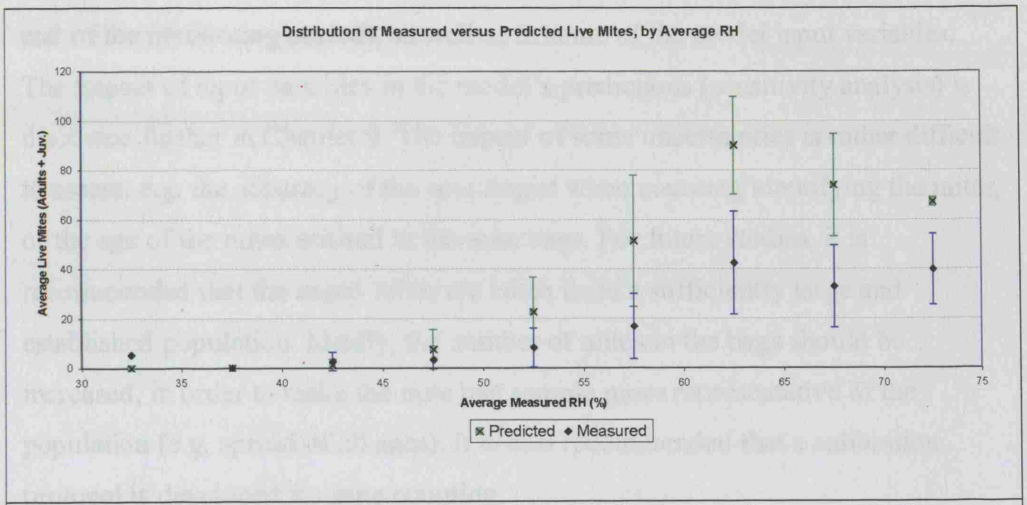


Fig 7.4.7 Distribution of predicted and measured live mites, by average RH measured in the correspondent mite bags.

7.5 Summary discussion

This section presented the mite bags results, and compared them with the predictions of the Popmite model (version 7d). The results confirm that the mite bags are an effective method, since population growth was observed under favourable hygrothermal conditions. The results also suggest that the mites in the bags thrived more if the average RH was greater than 55-60%, and the average temperature was 16-22 °C.

The comparison between measurements and predictions suggests that there is a moderate correlation between measured and predicted results (R-square value=0.58, juveniles+adults data) and that Popmite should be used for predicting the effect of hygrothermal conditions on *wild* DP mites, as opposed to laboratory-reared DP mites. Popmite is better at predicting juveniles than adults, over a 6-weeks period. However, Popmite 7d tends to over-predict juveniles numbers, particularly at high RHs. This suggests that in Popmite mites may be breeding too fast once the hygrothermal conditions are ideal, in comparison with the caged mite results. However, several sources of uncertainty exist in the measurements (e.g. loggers accuracy, mite counting and identification at the beginning and at the

[15] The Hobo H8 dataloggers used in the eight Series 1 beds had an accuracy of 0.7 ^{0}C for the temperature, and of 5% for the RH. The Type K thermocouples used for the four Series 2 beds had an accuracy of 1 $^{\circ}$C, while the RH sensors (Honeywell HIH-3610 Series) had an accuracy of 2% (although the RH sensors had been calibrated, under steady state conditions).

end of the monitoring period), as well as in some of the model input variables. The impact of input variables in the model's predictions (sensitivity analysis) is discussed further in Chapter 9. The impact of some uncertainties is rather difficult to assess: e.g. the accuracy of the acarologist when counting/identifying the mites, or the age of the mites utilised in the mite bags. For future studies, it is recommended that the caged mites are taken from a sufficiently large and established population. Ideally, the number of mites in the bags should be increased, in order to make the mite bag sample more representative of the population (e.g. spread of all ages). It is also recommended that a calibration protocol is developed for mite counting.

Although more work is required on Popmite in order to obtain improved predictions and reduce overpredictions, the Popmite predictions still reflect the measurements (Figure 7.3.8) and therefore Popmite 7d can be utilised in scenarios modelling, for assessing whether certain hygrothermal conditions generate more/less mites, *in comparison* with other conditions.

Chapter 7: References

Arlian LG and Veselica MM, (1981), Effect of temperature on the equilibrium body water mass in the mite *Dermatophagoides farinae*, *Physiology Zoology*, 54(4): 393-399.

Arlian LG, Neal JS, Morgan MS, Vyszenski-Moher DL, Rapp CM, Alexander AK, (2001), Reducing relative humidity is a practical way to control dust mites and their allergens in homes in temperate climates, *Journal of Allergy and Clinical Immunology*, 107(1): 99-104.

Biddulph P, Crowther D, Leung B, Wilkinson T, Hart B, Oreszczyn T, Pretlove S, Ridley I, Ucci M, (2007), Predicting the population dynamics of the house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle, *Experimental and Applied Acarology*, 41(1-2): 61-86.

Hart BJ, Crowther D, Wilkinson T, Biddulph P, Ucci M, Pretlove S, Ridley I, Oreszczyn T, (2007), Reproduction and development of laboratory and wild house dust mites (Acari: Pyroglyphidae) and their relationship to the natural dust ecosystem, *Journal of Medical Entomology,* 44(4): 568-574.

Pike AJ; Cunningham MJ; Lester PJ, (2005), Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) at constant and simultaneously fluctuating temperature and humidity conditions, *Journal Of Medical Entomology*, 42(3): 266-269.

CHAPTER 8:

COMPARISON OF FIELDWORK RESULTS WITH MPI PREDICTIONS

CHAPTER 8: COMPARISON OF FIELDWORK RESULTS WITH MPI PREDICTIONS

8.1 Introduction

This chapter discusses the comparison between fieldwork data (caged mites) and the predictions of the MPI model (Crowther *et al.*, 2006). The MPI model predicts the effect of steady-state hygrothermal conditions on house dust mite (DP) populations. The MPI output is the mite population index (MPI), such that 1.1 indicates 10% population growth and 0.9 indicates 10% population decline. The model was developed by carrying out laboratory experiments using laboratory-reared cultures of *Dermatophagoides pteronyssinus* (DP), feeding on an "artificial" diet (1:1 by weight, liver and yeast). The population change was observed for DP mites held in steady-state conditions at different combinations of temperature and RH over 21 days. The starting population was on average 1161 mites (eggs were not counted). From the laboratory results, a best-fit equation was derived which forms the basis of the MPI model. Further details on the MPI model are provided in Chapter 3. In this chapter, the predictions of the MPI model are compared with the results from the caged mites in the fieldwork study ("mite bags", see Chapter 4).

It should be pointed out that the mite bags were mostly developed for testing the Popmite model - i.e. for transient conditions. Some dissimilarities exist between the mite bags experiments, and the experiments on which the MPI model is based. Firstly, the mite bags were kept under transient conditions for 6 weeks, as opposed to steady-state conditions for 21 days. Secondly, the mites utilised in the mite bags for the Series 2 and Series 3 fieldwork studies were 'wild' mites, feeding on a natural diet (Hart *et al.*, 2007) – as opposed to laboratory-reared mites feeding on a lab-diet, as in the MPI experiments and in the Series 1 mite bags. Furthermore, the starting population in the mite bags is rather low (50 adult mites in Series 1, and 20 adult mites in Series 2 and 3), compared with the starting population in the MPI experiments (1161 mites, mix of all ages). Consequently, it was anticipated that some discrepancies would occur between MPI predictions and mite bag results, particularly for the Series 2 and 3 studies.

The next section (8.2) illustrates the methodology and the assumptions utilised for the comparison of the MPI predictions with the fieldwork data. The results are illustrated in section 8.3. The chapter ends with a summary discussion section (8.4).

8.2 Methodology and assumptions

This section illustrates the methodology and the assumptions utilised for the comparison of the MPI predictions with the fieldwork data.

In the fieldwork study each mite bag was monitored for a number of n days (on average 42 days), after which the final number of live mites in the bag was counted. However, the MPI output corresponds to the Mite Population Index after *21 days*, for given steady-state hygrothermal conditions. By assuming a constant rate of population growth, the predicted MPI value corresponding to n days (where the mites are exposed to steady-state conditions) can be calculated as follows:

$$MPI_n = (MPI)^{\frac{n}{21}} \qquad [8.1]$$

The predicted MPI can be compared with the "measured" MPI, which corresponds to the measured final number of live mites for each mite bag location (average of 3 mite bags), divided by the starting population in the mite bag (i.e. 50 mites for Series 1, 20 mites for Series 2 and 3).

The two main sources of uncertainties in the comparison between MPI predictions and the measurements are:

1. Uncertainties in the input hygrothermal conditions, which are dependent on the loggers' accuracy;
2. Uncertainties in counting the mites, both when the bags were initially made, and at the end of the monitoring period.

The first source of uncertainty is relatively easy to assess. In the fieldwork study, the hygrothermal conditions - to which the mite bags were exposed - had been monitored by one of the following logger types, depending on the mite bags

location (see Chapter 4): Hobos (www.onset.com)[1]; TinyTags (http://www.geminidataloggers.com/)[2]; Campbell Scientific datalogger CR23x (www.campbellsci.co.uk), with thermocouples Type K (RS Components, **rs**www.com)[3] and RH sensors Honeywell HIH-3610 Series (RS Components, **rs**www.com/)[4].

In order to estimate the total error due to inaccuracies in the temperature and RH readings, the Differential Sensitivity Analysis (DSA) method was utilised (Lomas and Eppel, 1992), which was reviewed in Chapter 2. For estimating the uncertainty associated with the hygrothermal inputs, the manufacturer's quoted accuracy was assumed to be equivalent to +/-2.33 x standard deviations, as already explained in Chapter5.

The other uncertainty – related to inaccuracies in mite counting – is much harder to assess. Since no "calibration" of the acarologist performing the counting had been carried out, the assessment of errors in mite counting could not be estimated. However, this inaccuracy might be partly taken into account since 3 replicates were used in each mite bags location. However, this does not exclude any systematic error in mite counting: for example, if the mite number is high, it might more difficult to count them.

In this section the methodology and assumptions utilised for the comparison of the MPI predictions with the fieldwork data were described. The next section illustrates the results of such comparison.

8.3 MPI predictions and fieldwork data: results

The results of the comparison between MPI predictions and fieldwork data are presented in this section.

[1] Hobo H8 Series. Temperature range: -20 to 70 ^{0}C; RH range: 25% to 95%. Temperature accuracy: +/- 0.7 ^{0}C at 21 ^{0}C. RH accuracy: +/- 5% over the range of 5 to 50 ^{0}C.
[2] TinyTag Ultra, Part Number 1500. Temperature range: -30 to 50 ^{0}C; RH range: 0 to 95%. Temperature accuracy: +/- 0.2 ^{0}C. RH accuracy: +/- 3%.
[3] Type K thermocouple, RS Components. Temperature range: -40 to 1200 ^{0}C. Temperature accuracy: +/- 1 ^{0}C.
[4] Honeywell HIH-3610 Series, RS Components. RH range: 0% to 90%. RH accuracy: +/- 2% at 25 ^{0}C. However, these sensors were calibrated using a Hobo H8, whose accuracy is utilised instead.

The starting population in the Series 1 mite bags consisted of 50 laboratory-reared mites, which is closer to the MPI original experiments than the mite bags utilised for the Series 2 or 3 studies. Consequently, it might be expected that the Series 1 results show a better correlation with the MPI predictions, than Series 2 and 3. However, very little correlation was found between MPI predictions and Series 1 results (Figure 8.1). This is most probably because of the small sample size of the Series 1 study. Furthermore, there was an insufficient variation in Series 1 fieldwork results: most mites were dead at the end of the monitoring period, since the mite bags were located in an unfavourable location (mattress top surface, under the chest area).

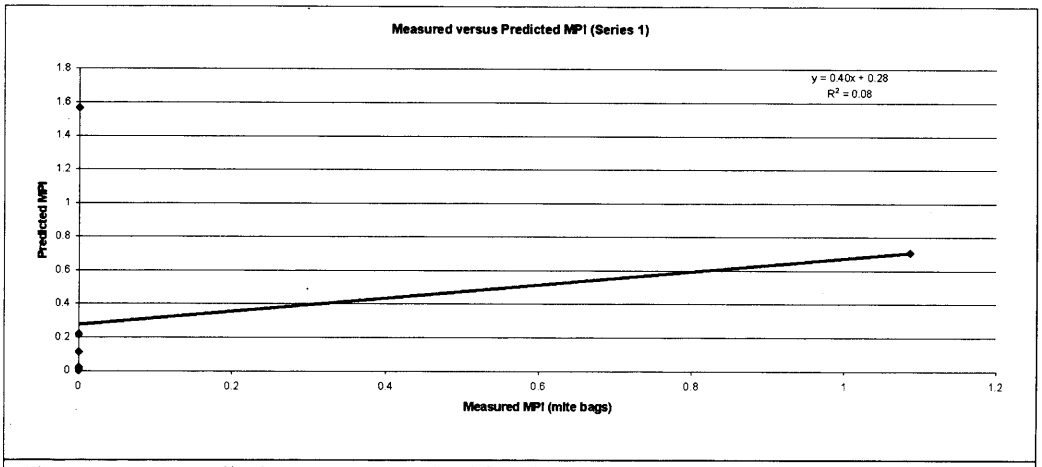


Fig 8.1 MPI predictions compared with mite bag measurements, for the Series 1 fieldwork study.

However, a better correlation between measurements and predictions was found when utilising the results from all the 3 fieldwork studies (Figure 8.2). This correlation did not differ when considering the results from Series 2 and 3 only.

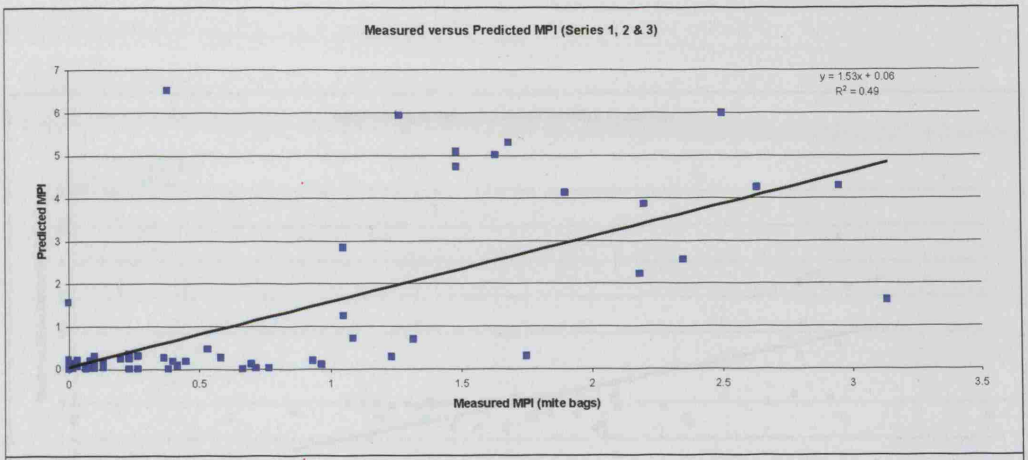


Fig 8.2 MPI predictions compared with mite bag measurements, from the Series 1, 2 and 3 fieldwork studies.

It should be noted that the R-squared value found for the MPI predictions against the measured results (0.49) is smaller than the R-squared value obtained for Popmite predictions from transient hygrothermal inputs (Popmite R-squared value: 0.58, see Chapter 7). This is somewhat expected, since the mite bags were kept under transient conditions. Both MPI and Popmite tend to over-predict by a factor of 1.5.

Figures 8.3 shows the average RH (measured in the mite bags) versus the difference between the predicted and the measured MPI. Figure 8.4 shows the difference between measurements and predictions, by the percentage of time the measured RH was above the Critical Equilibrium Humidity (CEH). The graphs reveals that for high average RHs and/or high amount of time the RH>CEH, the MPI model predicts the results less accurately than at low RHs, with a tendency for over-predictions. In mid range values, the model tends to under-predict the results.

Figure 8.5 shows the difference between measurements and predictions, by the average temperature measured in the mite bag. The graph reveals that when the temperature is between 19 and 23 °C, the model is less accurate in its predictions.

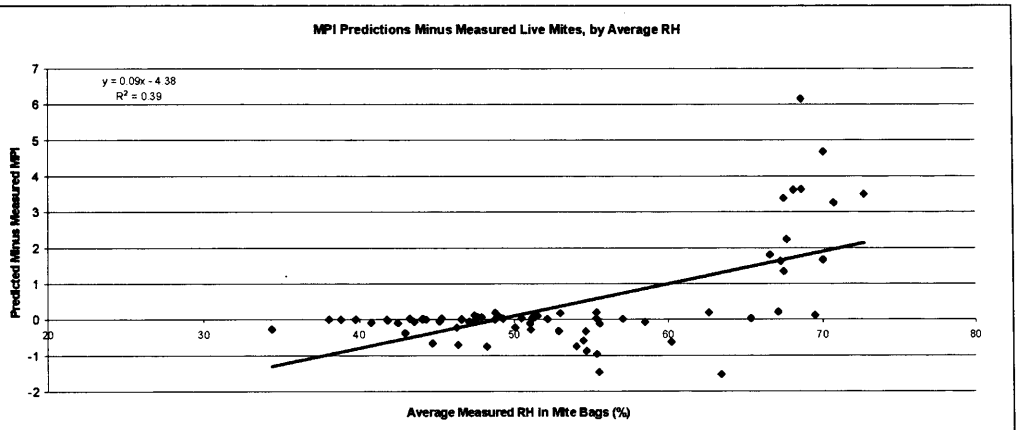


Fig 8.3 Difference between measured MPI in mite bags and predicted MPI, by average RH.

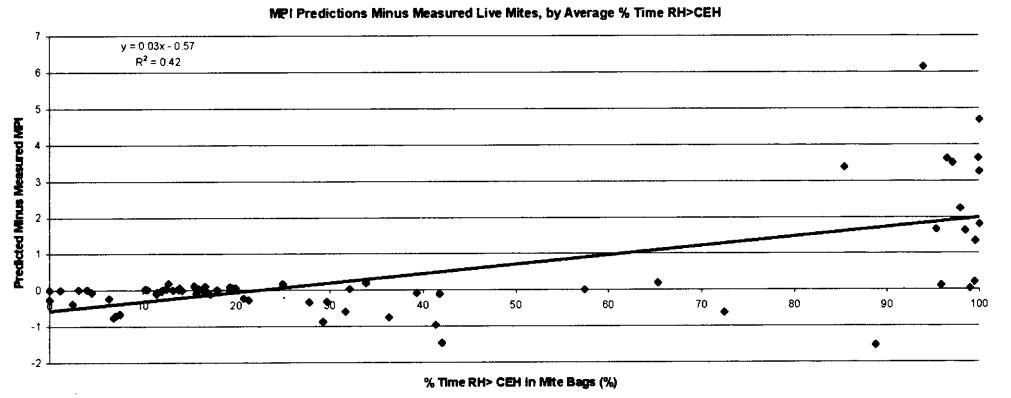


Fig 8.4 Difference between measured MPI in mite bags and predicted MPI, by percentage of time RH>CEH.

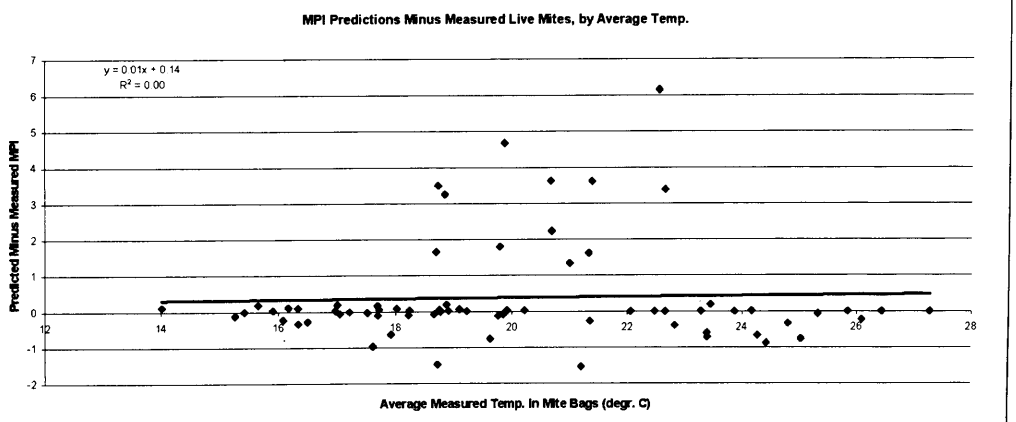


Fig 8.5 Difference between measured MPI in mite bags and predicted MPI, by average temperature.

Figure 8.6 shows the same data as Figure 8.2, but it includes the uncertainties due to inaccuracies in input hygrothermal conditions. The solid line represents where the data points would be, if a perfect fit was achieved between measurements and predictions.

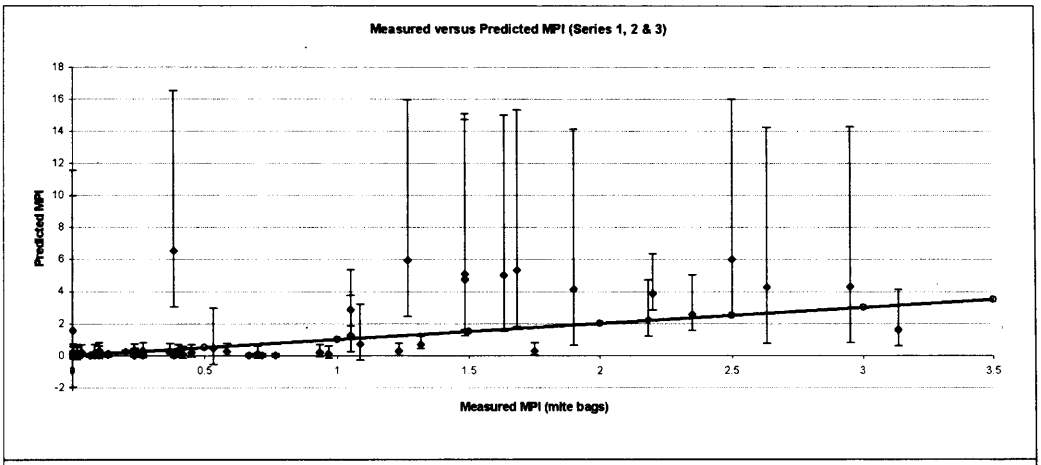


Fig 8.6 MPI predictions compared with mite bag measurements (Series 1, 2, and 3), including the uncertainties due to inaccuracies in input hygrothermal conditions.

The graph shows that when the measured MPI is greater than 1.5, the predicted MPI have a greater tendency to be higher than measured results. However, error bars are fairly large, and most bars cross the "perfect fit" line. When the measured MPI is between 0.5 and 1.5, some under-predictions occur, mostly with small error bars which do not cross the "perfect fit" line. In Figure 8.3 it should also be noted that the error bars are different for the various data points. This is partly the effect of the different sensors (and their accuracies) utilised for the fieldwork. However, this is also due to the base case hygrothermal conditions: if these are very unfavourable to mite growth, then small variations in temperature and RH do not lead to significant changes in MPI predictions. If on the other hand conditions are more favourable to mites, then even small hygrothermal changes can lead to noticeable differences in MPI predictions. Also, error bars *above* the base-case tend to be greater than those *below* base-case. This might suggest that the model has an intrinsic tendency for over-prediction.

Figure 8.7 shows the comparison between the MPI plot, and the *measured* population equilibrium. The graph is similar to the one published by Crowther *et al.* (2006) (Figure 3.4.1, Chapter 3). In Figure 8.7 the red line represents the MPI isogrowth curve where the MPI equals 1 (mite population stable). The heavy

dashed line is an estimation of the CEH for DP mites, based on published data for DF mites and 4 points for DP. The new element in the graph is represented by the blue line, which is the 'measured' isogrowth curve, where the mite bags measurements indicated a condition of population equilibrium (i.e. final mite count equal to initial number of mites in the bags). The black solid curves correspond to 'measured' population growth values of 2, 0.5 etc. The blue and the black curves were calculated as a contour plot of the measurement results presented in Figure 7.3.4 (Chapter 7). As already mentioned in the previous chapter, it should be highlighted that Figure 7.3.4 was based on rather large 'bins' and consequently has a rather wide uncertainty range. For example, each RH bin was 15% wide. Therefore, the contour plot (i.e. the blue line in Figure 8.7) could move by as much as 15%, once more data becomes available.

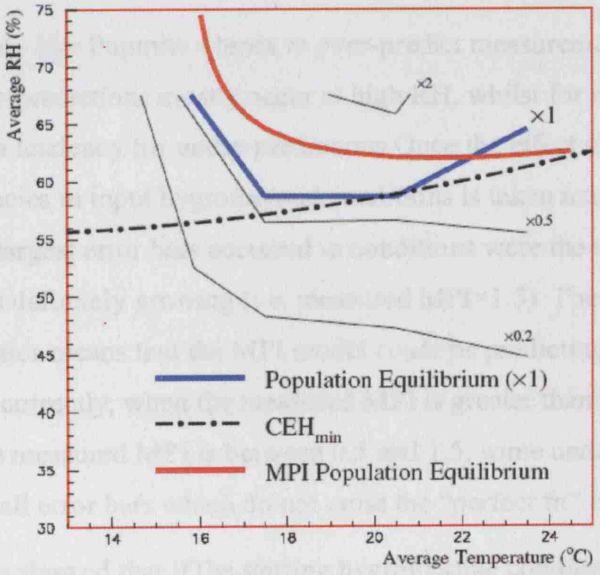


Fig 8.7 Comparison between the MPI isogrowth curve for population equilibrium (red curve), and the 'measured' isogrowth curve for population equilibrium (blue curve).

Considering all the uncertainties illustrated earlier, the graph shows a reassuring agreement between the MPI model – which was developed with laboratory-reared mites under laboratory steady-state conditions – and the mite bags results, which are based on caged wild mites exposed to real transient conditions. This also suggests that the mite bags are an effective method for exposing mite populations to a range of hygrothermal conditions.

The next section provides a summary discussion on the comparison between MPI predictions and mite bag results.

8.4 Summary discussion

This chapter discussed the comparison between MPI predictions and the results from the fieldwork studies. It was found that MPI predictions fit the field measurements less accurately than Popmite predictions (MPI R-squared value: 0.49; Popmite R-squared value: 0.58). This was somewhat expected, since the mite bags were kept under transient conditions, whilst MPI is a steady-state model. Furthermore, the experiments which formed the basis of the MPI model included a larger population of laboratory-reared mites, as opposed to a smaller population of *wild* mites on a "natural" diet (Series 2 and 3 mite bags).

The MPI model - like Popmite - tends to over-predict measurements by a factor of 1.5. These over-predictions mostly occur at high RH, whilst for mid-range values the model has a tendency for under-predictions.Once the effect of uncertainties due to inaccuracies in input hygrothermal conditions is taken into account, it was found that the largest error bars occurred in conditions were the mite bag population was definitely growing (i.e. measured MPI>1.5). The effect of these large uncertainties means that the MPI model *could* be predicting the measurements correctly, when the measured MPI is greater than 1.5. On the other hand, when the measured MPI is between 0.5 and 1.5, some under-predictions occur, with small error bars which do not cross the "perfect fit" line.

The results also showed that if the starting hygrothermal conditions are rather favourable to mite growth, even small changes in these initial conditions can lead to rather different MPI predictions, with a tendency for a larger MPI number (i.e. growth).

More conclusive results could have been obtained, if it had been possible to assess the errors in mite counting. Unfortunately, this is rather difficult to assess. For future studies, the development of a calibration methodology for the counting process is recommended.

Chapter 8: References

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

Hart BJ, Crowther D, Wilkinson T, Biddulph P, Ucci M, Pretlove S, Ridley I, Oreszczyn T, (2007), Reproduction and development of laboratory and wild house dust mites (Acari: Pyroglyphidae) and their relationship to the natural dust ecosystem, *Journal of Medical Entomology,* 44(4): 568-574.

Lomas and Eppel, (1992), Sensitivity analysis techniques for building thermal simulation programs, *Energy and Buildings*, 19: 21-44.

CHAPTER 9:

SENSITIVITY ANALYSIS

CHAPTER 9: SENSITIVITY ANALYSIS

9.1 Introduction

This chapter discusses the sensitivity of the hygrothermal and population models to their input variables. For each model, the main aims of the sensitivity analysis are:

1. Help identifying those input variables which need to be known with greater certainty, in order to obtain more accurate predictions;
2. Help identifying those input variables which have the greatest impact on mite growth/decline, and hence help devise appropriate control strategies.

For each model, the sensitivity analysis was performed by changing one at a time each input variable by +/- 10%, from a base-case value. The variables resulting in greater changes (from base case) in predictions are those to which the model is most sensitive. A 10% change from base-case was considered a reasonably realistic figure. However, it could be argued that the effects of a percentage change from base-case values also depend on the initial conditions to which such changes are applied. For example, a 10% change in temperature corresponds to 2 °C if the average base-case temperature is 20 °C, but if the latter is 15 °C, then the 10% change corresponds to a smaller reduction (in absolute terms) of 1.5 °C. However, the sensitivity analysis primarily aims to assess which variables have the greatest impact on predictions. In order to do so, percentage changes – rather than absolute changes[1] – have to be considered, since the various input variables have different units. Furthermore, in the Popmite model some input variables are hypothetical: i.e. they do not correspond to commonly used units. Therefore, in this case it would be difficult to identify by how much (in absolute terms) such input variables should be changed.

Since an effort was made to consider the effect of changes in input variables independently, the base-case RH was not modified as a result of the +/- 10% changes in input temperatures (and vice-versa). However, it should be highlighted that in real buildings a change in temperature is usually accompanied by a change in RH – unless a change in vapour pressure also occurs.

[1] One could, for example, examine the changes in predictions due to changes in temperature inputs of +/-1 °C.

The sensitivity analysis was performed for the models: Lectus (section 9.2), MPI (section 9.3) and Popmite (section 9.4). The chapter ends with a summary discussion (section 9.5). It was considered unnecessary to carry out a sensitivity analysis for the BED model, since this had already been carried out by Pretlove *et al.* (2005). In particular, Pretlove *et al.* carried out a sensitivity analysis of the BED model, in combination with the Condensation Targeter model (Oreszczyn and Pretlove, 1999), which predicts bedroom monthly conditions and can therefore be used in conjunction with the BED model. The authors found that the RH in the bed core is mostly dependant on parameters affecting room conditions, rather than parameters specific to the BED model. After room conditions, the BED-specific parameters to which the RH predictions are most sensitive were: the number of hours the bed is occupied and the skin temperature (Fig 9.1.1). The temperature predictions for the bed core are affected by: room temperature, skin temperature and number of hours the bed is occupied.

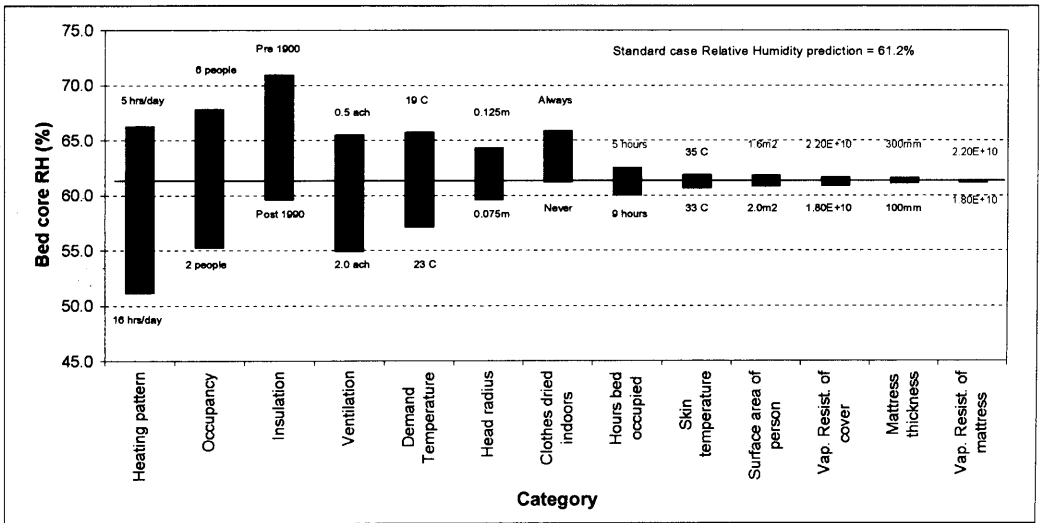


Fig 9.1.1 Sensitivity analysis of the BED model (Pretlove *et al.*, 2005)

The following section discusses the sensitivity analysis of the other bed hygrothermal model Lectus.

9.2 Lectus: sensitivity analysis

This section discusses the sensitivity analysis of the bed hygrothermal model Lectus (Ridley *et al.*, submitted). The model uses as main inputs the hourly temperature and relative humidity of the bedroom, the properties of the mattress

materials, and the boundary conditions on the surfaces of the mattress. The model splits the bed into a flexible user-defined three-dimensional grid, which can include multiple layers of different materials. Subsequently, predictions of hourly temperature and relative humidity for each mattress cells are provided (see Chapter 3).

As already mentioned in the introduction, the sensitivity analysis of the Lectus model was performed by changing one at a time each input variable by +/- 10%, from a base-case value. The base-case mattress was a 15 cm thick single bed made of homogeneous material, whose properties were taken from different published sources to represent a foam mattress. The input room conditions were those measured in one of the fieldwork beds (Series 2, bed 2.1). For the boundary conditions, these were based on the Lectus model for boundary conditions. These assume that at all times other than when the bed is occupied and on all surfaces other than the top, the boundary conditions are the same as the room. When the bed is occupied, the top surface conditions in each mattress zone are those illustrated in Figure 9.2.1. In the sensitivity analysis it was assumed that the base-case bed was occupied for 8 hours per day.

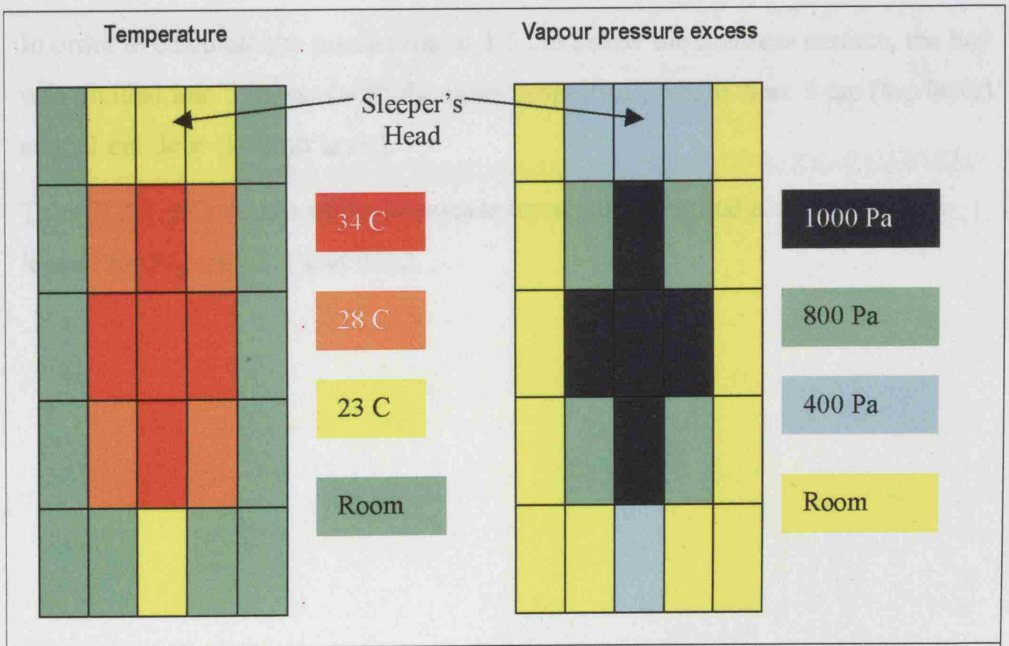


Fig. 9.2.1 Top of the mattress boundary conditions in Lectus, for an occupied sprung mattress.

Lectus predicts hygrothermal conditions in a number of cells, into which the mattress is split. However, it was decided to focus the sensitivity analysis on the predictions for a specific cell, which corresponds to the area under the chest (when the bed is occupied) and whose centre is at 1.5 cm from the top surface of the mattress. This cell was selected for two reasons:

a) Preliminary measurements have shown that the mattress area at 1-2 cm below the top mattress surface may be most favourable for mite colonisation - provided physical access is achievable at these other locations and food is accessible (Pretlove *et al.*, 2005; Ridley *et al.*, submitted).

b) In Lectus, when the bed is unoccupied the boundary conditions for the mattress are identical to room conditions. Therefore, after some time the conditions within the mattress are the same as room conditions, regardless of changes in any other input variables (see Chapter 5). However, when the bed is occupied, the cells underneath the chest area are subjected to the greatest hygrothermal gradient (from the top to the bottom surface). Therefore, the cell underneath the chest area is potentially the most sensitive to changes in input parameters.

In order to calculate the predictions at 1.5 cm below the mattress surface, the bed was divided into 2 layers (with the same properties), which were 3 cm (top layer) and 12 cm deep (bottom layer).

Table 9.2.1 give details of the base-case input variables, and it also provides a legend for Figure 9.2.1 and 9.2.2.

Table 9.2.1 Base values for the Lectus input variables, and legend for Figure 9.2.1 and 9.2.2.

Input Parameter	Base value	Legend for Fig 9.2.1-2	
		+10%	-10%
Density	36 Kg/m^3	1	2
Thermal Conductivity	0.06 W/mK	3	4
Heat Capacity	850 J/kgK	5	6
Vapour Permeability	8.70E-09 kg/msPa	7	8
Moisture Capacity	2.00E-05 kg/kgPa	9	10
Thickness	0.15 m	11	12
Time in Bed	8 hours	13	14
Half Life*	30 minutes	15	16
Boundary Conditions, Temp.	(see Fig 9.2.1)	17	18
Boundary Conditions, V.P.	(see Fig 9.2.1)	19	20
Calculations time-step#	20 seconds	21	22
Room Conditions, Temp.^	23.4 °C	23	24
Room Conditions, RH^	54.5%	25	26

*Time it takes for the difference between the mattress top surface conditions and the room conditions to be halved, once the bed is vacated. This determines how quickly the boundary conditions go back to equilibrium with room conditions, once the bed is vacated.
#It determines the number of iterations for the calculations. This value has to be greater than the stability criterion, which is dependent on the mattress materials' properties.
^ Average Monitored Room Conditions, Bed 2.1

Figures 9.2.1 and 9.2.2 show the effect of changes on the temperature and RH predictions, due to changes in input parameters, over a 48 hours period. The legend for the input parameters in Figure 9.2.1 and 9.2.2 is given in the table above (Table 9.2.1). The graphs show that when the bed is not occupied, the only input variables whose changes modify base-case predictions are the room conditions (series 23 and 24 for the temperature in Fig 9.2.1, and series 25 and 26 for the RH in Fig 9.2.2). This is not surprising, since the boundary conditions are the same as room conditions, when the bed is unoccupied. When the bed is occupied, the input *temperature* boundary conditions have the greatest impact on predictions (series 17 and 18 in the graphs), followed by changes in room conditions (series 23 to 26 in the graphs), as well as in the *vapour pressure excess* boundary conditions, for the RH in Figure 9.2.2 (series 19 to 20 in the graphs).

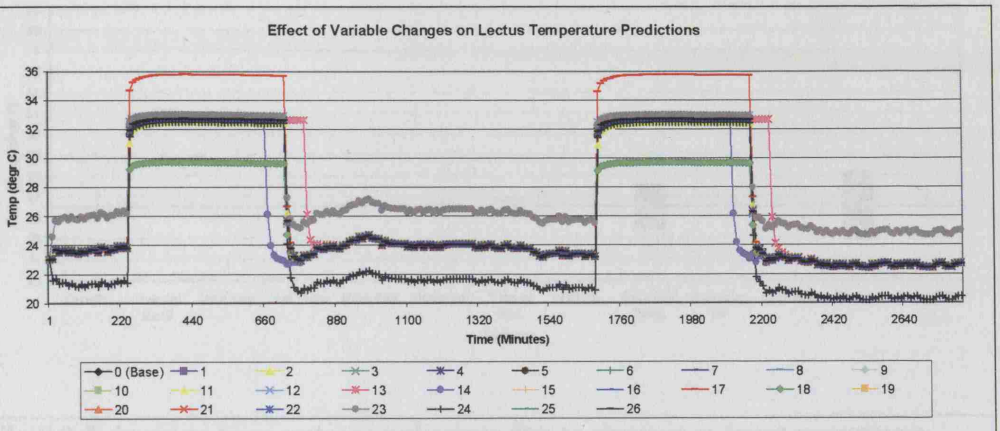


Fig 9.2.1 Effect of changes in input variable on Lectus temperature predictions (for a legend to the series names, refer to Table 9.2.1).

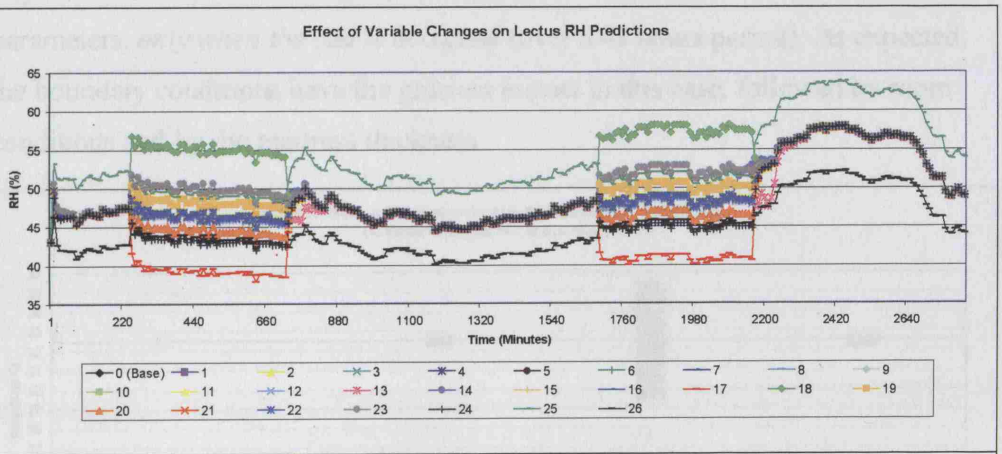


Fig 9.2.2 Effect of changes in input variable on Lectus RH predictions (for a legend to the series names, refer to Table 9.2.1).

It may be helpful to present the results in terms of average predictions (over 48 hours). Figure 9.2.3 shows the average temperature predictions due to changes in input parameters, over 48 hours. The graph shows that in most cases changes in input variable do not result in significant changes from base case predictions. However changes in room temperatures have a significant impact on changes in predictions, followed by changes in temperature boundary conditions, time in bed, and by the mattress thickness.

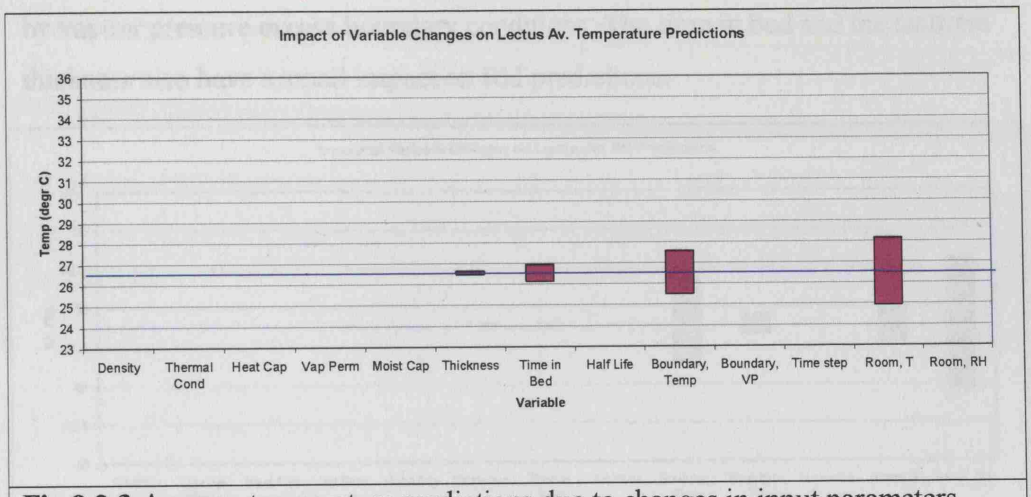


Fig 9.2.3 Average temperature predictions due to changes in input parameters, over 48 hours.

Figure 9.2.4 shows the average temperature predictions due to changes in input parameters, *only when the bed is occupied* (over a 48 hours period). As expected, the boundary conditions have the greatest impact in this case, followed by room conditions and by the mattress thickness.

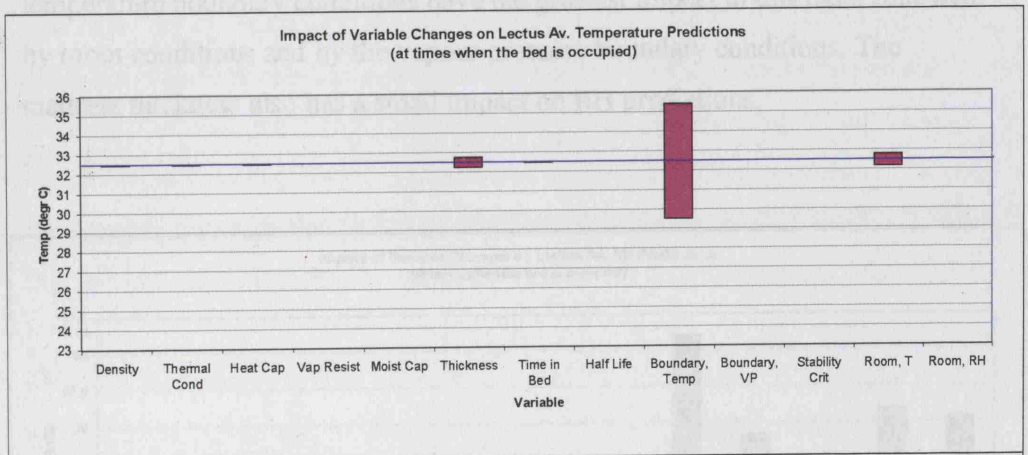


Fig 9.2.4 Average temperature predictions due to changes in input parameters, *only when the bed is occupied*, over 48 hours.

Figure 9.2.5 shows the average RH predictions, due to changes in input parameters, over 48 hours. The graph shows that in most cases changes in input variable do not result in significant changes from base case predictions. However changes in room RH have a significant impact on changes in predictions, followed by changes in temperature boundary conditions, room temperature and

by vapour pressure excess boundary conditions. The time in bed and the mattress thickness also have a small impact on RH predictions.

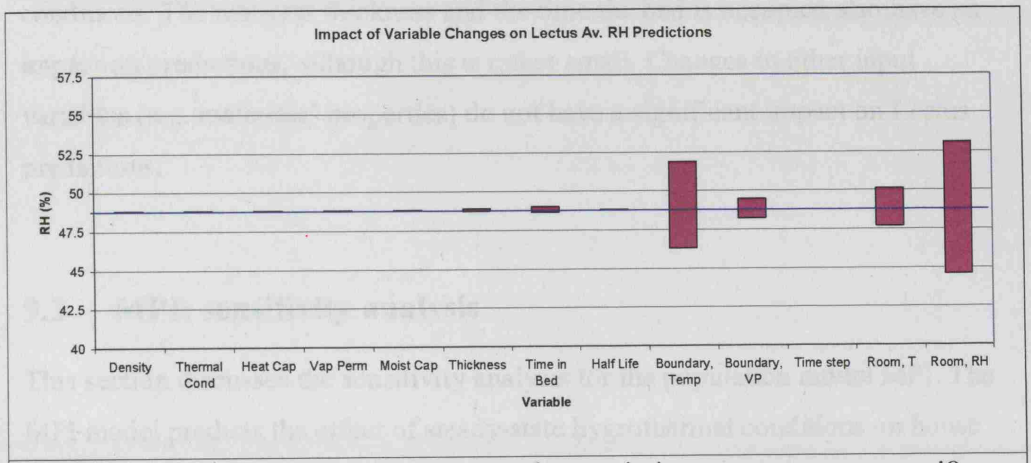


Fig 9.2.5 Average RH predictions due to changes in input parameters, over 48 hours.

Figure 9.2.6 shows the average RH predictions due to changes in input parameters, *only when the bed is occupied* (over a 48 hours period). The temperature boundary conditions have the greatest impact in this case, followed by room conditions and by the vapour pressure boundary conditions. The mattress thickness also has a small impact on RH predictions.

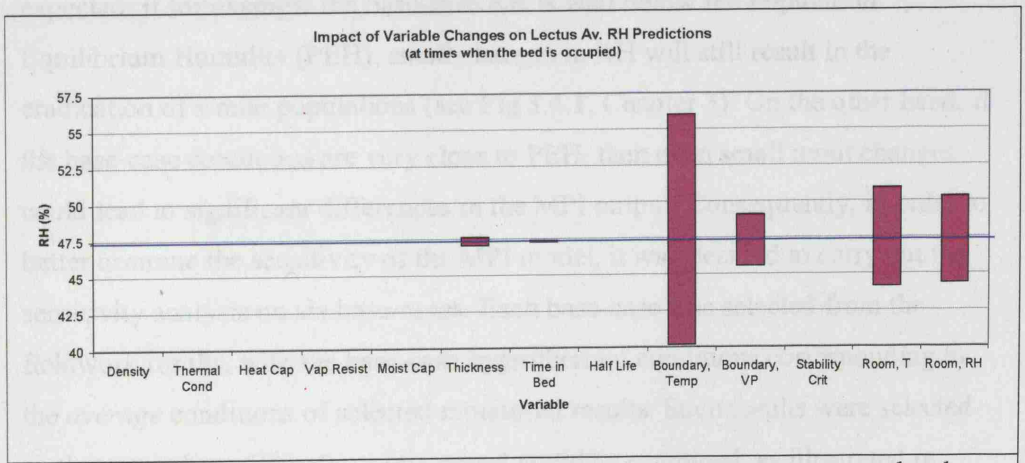


Fig 9.2.6 Average RH predictions due to changes in input parameters, *only when the bed is occupied*, over 48 hours.

This section discussed the sensitivity analysis of the Lectus model. The results show that the model is most sensitive to the room conditions and to the boundary conditions. The mattress thickness and the time the bed is occupied also have an impact on predictions, although this is rather small. Changes in other input variables (e.g. materials' properties) do not have a significant impact on Lectus predictions.

9.3 MPI: sensitivity analysis

This section discusses the sensitivity analysis for the population model MPI. The MPI model predicts the effect of steady-state hygrothermal conditions on house dust mite (DP) populations (Crowther *et al.*, 2006). The MPI output is the mite population index (MPI), such that 1.1 indicates 10% population growth and 0.9 indicates 10% population decline. The only input variables for the MPI model are the steady-state temperature and RH to which the mites are exposed.

As already mentioned in the introduction, the MPI sensitivity analysis was performed by changing one at a time each input variable by +/- 10%, from a base-case value. However, from the error analysis carried out in Chapter 8, it emerged that changes in input variables had a very different impact on changes in the MPI output, depending on the base-case hygrothermal conditions. This is expected: if for example the base-case RH is well below the Population Equilibrium Humidity (PEH), small changes in RH will still result in the eradication of a mite populations (see Fig 3.4.1, Chapter 3). On the other hand, if the base-case conditions are very close to PEH, then even small input changes could lead to significant differences in the MPI output. Consequently, in order to better examine the sensitivity of the MPI model, it was decided to carry out the sensitivity analysis on six base-cases. Each base-case was selected from the fieldwork results, with the base-case hygrothermal conditions corresponding to the *average* conditions of selected monitored results. Such results were selected so that a number of situations (six cases) could be examined, as illustrated in Table 9.3.1 - which summaries the average hygrothermal conditions of the selected 6 cases, as well as the overall average conditions for the total of 78 cases. The "high" and "low" values utilised to identify the cases in Table 9.3.1

were selected in relation to the average measured conditions found *overall* in the 78 cases monitored for the fieldwork study.

Table 9.3.1 Average hygrothermal characteristics and predicted MPI of selected base-cases and of the overall 78 fieldwork cases.

	Av. Temp. (°C)	Av. RH (%)	Av. CEH (%)	Base MPI*
Case 1 (Low T and RH)	18.2	40.8	55.6	0.14
Case 2 (High T and RH)	22.6	68.6	56.3	2.50
Case 3 (Low T and High RH)	18.7	72.7	55.6	2.40
Case 4 (High T and Low RH)	21.4	34.4	56.0	0.02
Case 5 (High T and RH close to CEH)	23.3	57.0	56.4	0.50
Case 6 (Low T and RH close to CEH)	18.7	55.5	55.6	0.55
Overall average of 78 cases	20.4	53.2	56.0	0.66

*Predicted

Based on the average hygrothermal conditions of the six selected hygrothermal base-cases illustrated in the Table above, the 10% change in base-case hygrothermal conditions corresponds to a variation of 1.8-2.3 °C in temperature, and of 3-7% in RH. Figure 9.3.1 shows the variation in MPI predictions for each selected base-case, by changes in temperature and in RH (+/- 10%).

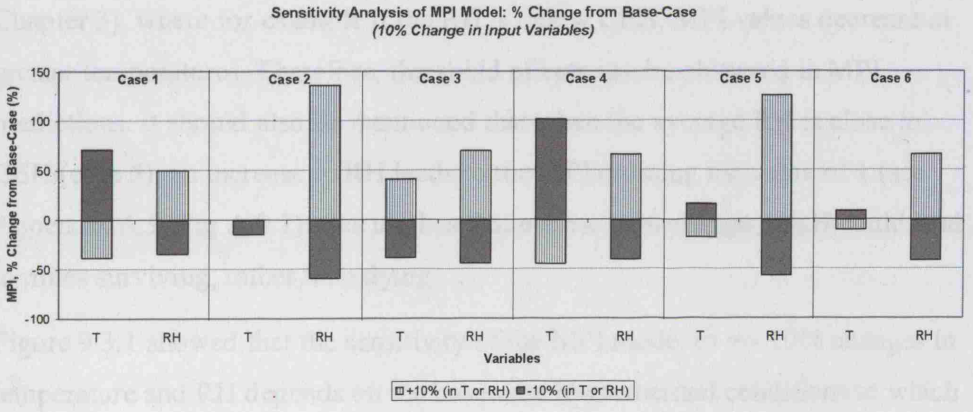


Fig 9.3.1 Percentage change from base-case MPI predictions, due to +/- 10% changes in temperature or RH, for the 6 selected base-cases (see Table 9.3.1).

Figure 9.3.1 shows that, for example, in the low temperature and low RH case (case 1, left hand side of the graph), a 10% reduction in temperature (corresponding to the data series with dots) leads to a 71% increase in the MPI, whilst a 10% increase in temperature (corresponding to the data series with lines) leads to a 39% reduction in the MPI. On the other hand, a 10% reduction in RH (corresponding to the data series with dots) leads to a 35% reduction in the MPI,

whilst a 10% increase in RH (corresponding to the data series with lines) leads to a 50% increase in the MPI. The graph shows that the sensitivity of the MPI model to changes in temperature or RH is dependent on the base-case hygrothermal conditions. The graph also shows that RH and MPI are always positively correlated (i.e. an increase in RH always leads to a increase in MPI, and vice-versa), whilst temperature and MPI are not consistently positively or negatively correlated. For example, in case 1, 5 and 6, a 10% reduction in temperature results in an increase in the MPI predictions, whilst in case 2 and 3 the reduction in temperature results in a reduction in the MPI prediction.

The graph also shows that in most cases the MPI model is more sensitive to 10% changes in RH than in temperature, except in case 1 and 4 – where there was a low base-case RH. However, in most cases an increase in temperature leads to a *reduction* in predicted MPI (cases: 1, 4, 5, 6), except in case 3 - where an increase in temperature leads to an increase of predicted MPI. This phenomena can be explained by considering the MPI graph representing the isogrowth MPI lines corresponding to combinations of hygrothermal conditions (Figure 3.4.1, Chapter 3), where for example if the RH is below CEH, MPI values decrease at greater temperatures. Therefore, threshold effects can be observed in MPI predictions. It should also be mentioned that when the average RH is close to CEH (case 5), an increase in RH leads to the MPI crossing the value of 1 (see Appendix A.9, Fig A.9.1): this implies that even a 10% change in RH could lead to mites surviving, rather than dying.

Figure 9.3.1 showed that the sensitivity of the MPI model to +/- 10% changes in temperature and RH depends on the base-case hygrothermal conditions to which such changes are applied. It could be argued that this phenomenon might be partly due to the fact that in absolute terms the size of the change is dependent on the base-case conditions. For example, if the base-case temperature is 20 °C, then a 10% change is 2 °C, which would be a bigger change (in absolute terms) than the case when for example the base-case temperature is 15 °C. Figure 9.3.2 shows the MPI sensitivity analysis results, where a fixed change in temperature and RH was applied to base-case input hygrothermal conditions. In particular, in each of the 6 input hygrothermal cases illustrated in Table 9.3.1, a +/- 1 °C change was applied for the temperature, and a +/- 5% was applied for the RH.

The graph shows that even when applying a fixed change in temperature or RH, the sensitivity of the MPI model changes, depending on the base-case conditions to which such changes are applied.

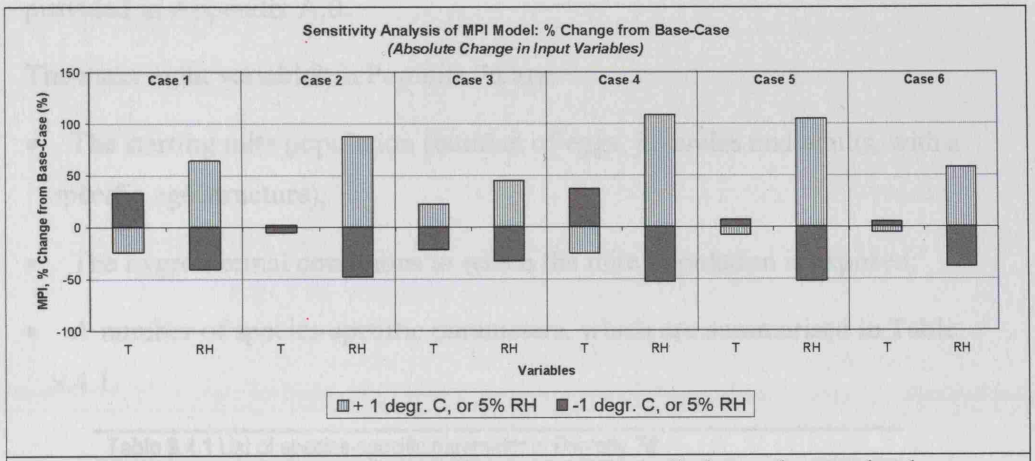


Fig 9.3.2 Percentage change from base-case MPI predictions, due to absolute changes in temperature (+/- 1 °C) or RH (+/- 5 %), for the 6 selected base-cases (see Table 9.3.1).

This section discussed the sensitivity of the MPI model. The results indicate that the sensitivity of the MPI model to changes in temperature or RH is dependent on the initial (base-case) hygrothermal conditions. Since threshold effects can be observed in the MPI predictions, it is not possible to quantify a *typical* change in MPI predictions, due to a given change in hygrothermal conditions. In most cases, the model is more sensitive to changes in RH, than to changes in temperature. However, depending on the base-case conditions and on the size of the change, changes in temperature can be important as well - particularly at low base-case RHs. Depending on the initial hygrothermal conditions (e.g. RH close to CEH), even a 10% change in these conditions might lead to a change in MPI output, such that the prediction of mite population *decline* is changed into a prediction of mite population *growth* (or vice-versa).

9.4 Popmite: sensitivity analysis

This section discusses the sensitivity analysis of the population model Popmite (version 7d). The Popmite 7d model predicts the effect of transient hygrothermal conditions on a population of DP mites, with a given population structure (i.e.

age and life cycle). A paper on Popmite 7d is being prepared by Biddulph *et al.* (see Chapter 3 for further information), although a paper introducing some of the concepts utilised in Popmite has been published (Biddulph *et al.*, 2007), and is provided in Appendix A.0.

The main input variables in Popmite 7d are:

- The starting mite population (number of eggs, juveniles and adults, with a specific age structure);
- The hygrothermal conditions to which the mite population is exposed;
- A number of species-specific parameters, which are summarised in Table 9.4.1.

Table 9.4.1 List of species-specific parameter in Popmite 7d

Parameter Name	Base Value	Units	Abbreviation
Fast Up Water Gain Rate	0.5	µg/hour	Fast Up
Slow Down Water Loss Rate	0.0012	µg/hour	Slow Down
Fast Threshold for Eating	1	%	Fast Thr.
Female Eating Rate	0.22	food units*/hour	Eat. Rate
Egg Food Cost	100	food units*/hour	Egg Cost
Maximum Food Intake	200	food units*/hour	Max Food
Egg Lay Total Water Threshold	70	%	Egg Thr.
Adult Female Population	50	%	Fem. Ratio
Water Slow Compartment Egg	0.5	µg	Slow Egg
Water Slow Compartment Adult	2.95	µg	Slow Ad.
Water Fast Compartment Adult	1.34	µg	Fast Ad.
Water Death Threshold	48	%	Wat. Death
Base Mortality Rate	15	%	Base Mort.

* Hypothetical

The DP species-specific parameters utilised in Popmite 7d were identified by the model developer (Dr Biddulph) from published values, or estimated on the basis of various sources on other species. However, for some of these parameters (e.g. female eating rate) very little published information is available and a hypothetical value was selected. As already mentioned in the introduction, each input variable in Popmite was increased and decreased by 10% (from a starting base-case), in order to assess the sensitivity of the model to its input variables.

As in the MPI model, it was anticipated that Popmite predictions would be affected by the base-case input hygrothermal conditions, for example because of the threshold effect represented by the temperature-dependent Critical Equilibrium Humidity (CEH). Consequently, it was decided to carry out a

sensitivity analysis on four *base-case input hygrothermal conditions*, which were selected from the fieldwork data. In particular, from the fieldwork data four input hygrothermal files were selected as follows:

a) One case where the measured final live mite count was the highest, with a measured high average RH, and with a high percentage of time where the monitored RH was above CEH. This case is designated as "High RH".

b) One case where the measured final live mite count was nil, with a measured low average RH, and with the lowest percentage of time the monitored RH was above CEH. This case was designated as "Low RH".

c) One case where the measured final live mite count and the hygrothermal conditions were closest to the average corresponding measures for all fieldwork cases. This case is designated as "Near CEH", since the RH was closest to the Critical Equilibrium Humidity, compared with the other cases.

d) One case with high measured average RH and high percentage of time RH>CEH, but with low measured average temperature. This case is designated as "Low Temperature".

The characteristics of each case are summarised in Table 9.4.2. The +/- 10% changes in hygrothermal conditions from base-case were carried out on the monitored transient data. It should be mentioned that in the original fieldwork study, the mite bags were monitored for slightly different lengths of time. However, for the purposes of this sensitivity analysis, the 4 hygrothermal base-cases were all utilised in Popmite with the same amount of time (974 hours; 40.6 days).

Table 9.4.2 Characteristics of the 4 hygrothermal input base-cases (measured values)

	Hygrothermal Input Base-Case Name			
	Low RH	*High RH*	*Low Temp.*	*Near CEH*
Average Monitored Temp. (°C)	22.7	21.2	14.0	19.9
Average Monitored RH (%)	38.0	63.4	69.5	58.5
% time RH > CEH (%)	0	88.8	95.8	39.3
Average Difference: RH-CEH (%)	-18.3	7.2	14.2	-1.6
Final count in mite bags: adults	0	19	4	4
Final count in mite bags: juveniles	0	44	1	7

From Table 9.4.2 it can be observed that the base-case "Low Temperature" had the higher RH and percentage of time RH>CEH, but it did not have the highest mite count, due to low temperatures. Also, the base-case named "Near CEH" has a small average difference between RH and CEH, suggesting that the hygrothermal conditions were very close to CEH.

For each of the 4 hygrothermal input base-cases, the Popmite input parameters (incl. temperature and RH) were increased/decreased one at a time by 10% from the base-case. The starting mite population was assumed as 20 adults (10 males, 10 females), with a spread of all ages[2]. Figures 9.4.1-3 illustrate the results of the sensitivity analysis, with a focus on adult mite predictions. The results for other output types (i.e. juveniles and eggs) are illustrated in Appendix A.9, as well as in Figure 9.4.4-7 (for the impact of changes in hygrothermal conditions). It should also be mentioned that the results for the hygrothermal base-case "Low RH" are not illustrated in Fig. 9.4.1-3, since no changes in predictions were observed, for +/- 10% changes in input variables.

[2] Some discrepancies occur between the live mite measurements in Table 9.4.2, and the live mites base-case predictions (e.g. see Case "Average"). However, such discrepancies do not affect the results of this sensitivity analysis.

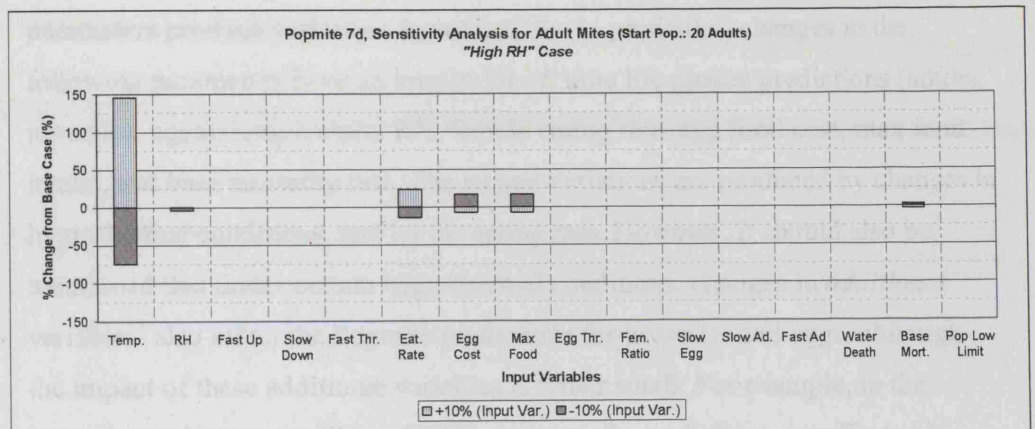


Fig 9.4.1

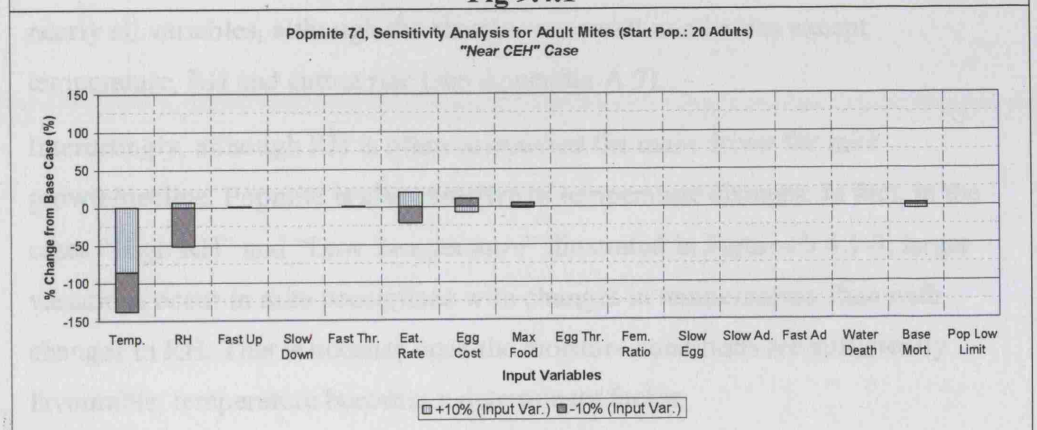


Fig 9.4.2

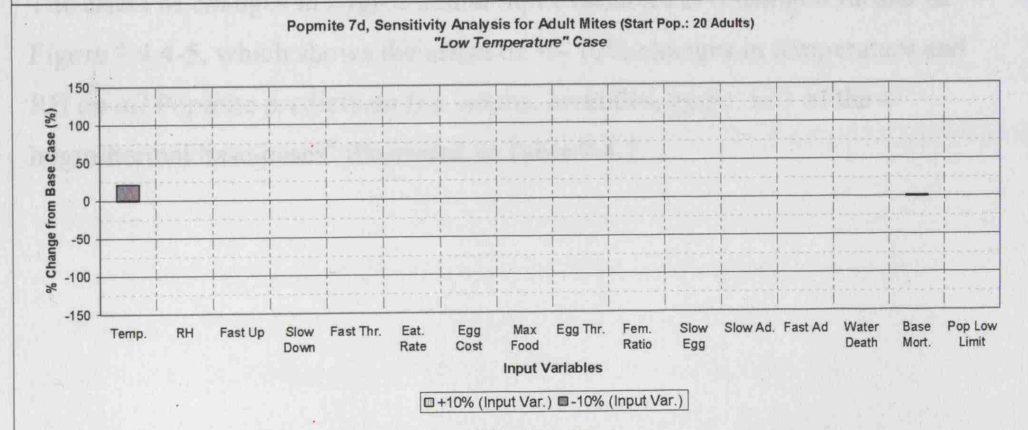


Fig 9.4.3

Fig 9.4.1-3 Popmite, mite adult predictions: sensitivity to +/- 10% changes in input variables, for 3 different input hygrothermal cases (see Table 9.4.2).

The results suggest that the impact of changes in input parameters is dependent on the input hygrothermal base-case. For example, in conditions of very low RH, the Popmite predictions are always nil, regardless of 10% changes in input parameters. However, for the other 3 hygrothermal base-cases, changes in input

parameters produce variations in predictions. In particular, changes in the following parameters have an impact for *all* mite life phases predictions (adults, juveniles, eggs): temperature, RH, female eating rate, egg food cost, max food intake, and base mortality rate. The largest variations are produced by changes in hygrothermal conditions, and by the eating rate. However, it should also be mentioned that under certain hygrothermal conditions, changes in *additional* variables[3] also affect the Popmite predictions for juveniles and eggs, although the impact of these additional variables is rather small. For example, in the hygrothermal base-case "Near CEH", the juvenile predictions are affected by nearly all variables, although the changes are small in all cases except temperature, RH and eating rate (see Appendix A.9).

Interestingly, although RH is often considered the main driver for mite growth/decline, Popmite is also sensitive to temperature changes. In fact, in the cases "High RH" and "Low Temperature" illustrated in Figures 9.4.1-3, larger variations occur in mite predictions with changes in temperatures, than with changes in RH. This is because once the moisture conditions are sufficiently favourable, temperature becomes a determinant factor.

The effect of changes in hygrothermal input variables is illustrated further in Figure 9.4.4-5, which shows the effect of +/- 10% changes in temperature and RH on *all* Popmite predictions (i.e. adults, juveniles, eggs), in 3 of the 4 hygrothermal base-cases[4] illustrated in Table 9.4.1.

[3] Other than those previously identified as the main variables to which Popmite is sensitive: temperature, RH, eating rate, and to a lesser extent egg cost, max food intake and mortality rate.
[4] As previously mentioned, for the hygrothermal base-case "Low RH", no changes were observed in Popmite predictions for +/- 10% changes in any input variable.

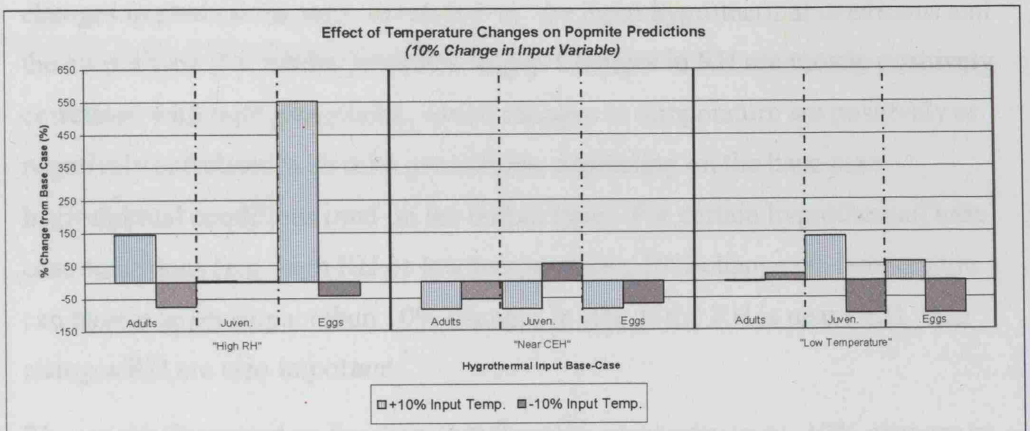


Fig 9.4.4

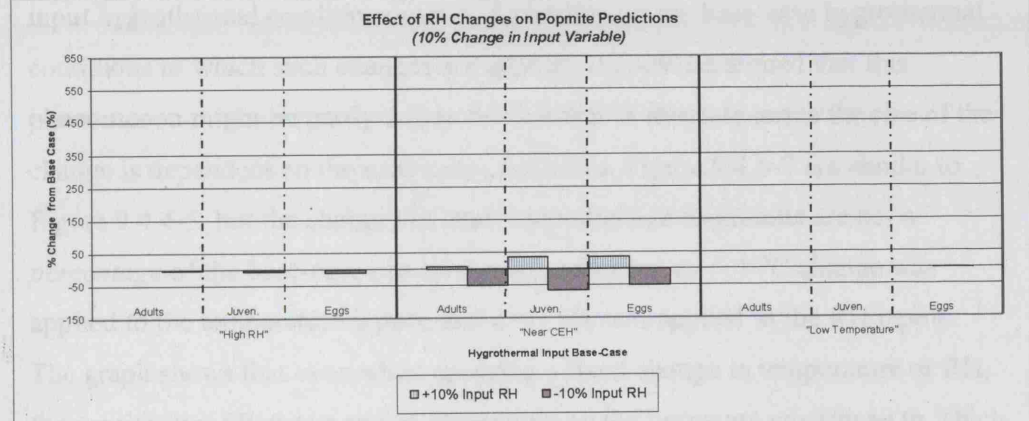


Fig 9.4.5

Fig 9.4.4-5 Effect of changes (+/- 10%) in temperature or RH on Popmite predictions. Starting population: 20 adults, spread of all ages.

In Figure 9.4.4-5, on the x-axis the 3 hygrothermal base-cases are separated by a solid line, and each base-case is separated further for predictions in: adults, juveniles or eggs (separated by a dashed line). For each of the cases illustrated on the x-axis, the percentage change from base-case predictions due to +/- 10% changes in temperature (or RH) is presented on the y-axis. For example, Figure 9.4.4 shows that in the hygrothermal base-case "High RH", there was nearly a 150% increase from base-case predictions in adults (first column from the left), due to a 10% increase in temperature, whilst a 74% reduction in base-case adult predictions (second column from left) could be observed, for a 10% reduction in temperature.

The graphs show that the size and the direction (i.e. increase or decrease) of the changes in predictions vary, in relation to: the input hygrothermal conditions and the output type (i.e. adults, juveniles, eggs). Changes in RH are mostly positively correlated with mite predictions, whilst changes in temperature are positively or negatively correlated with mite predictions, depending on the base-case hygrothermal conditions (and on the output type). For certain hygrothermal base-case conditions (e.g. high RH or low temperature), 10% changes in temperature can have a larger impact than 10% changes in RH. If the RH is near CEH, then changes RH are also important.

The graphs illustrated so far show that Popmite sensitivity to +/- 10% changes in input hygrothermal conditions varies, depending on the base-case hygrothermal conditions to which such changes are applied. It could be argued that this phenomenon might be partly due to the fact that in absolute terms the size of the change is dependent on the base-case conditions. Figure 9.4.6-7 are similar to Figure 9.4.4-5, but the changes in input hygrothermal conditions are not a *percentage* of the base-case conditions. In particular, a +/- 1 °C change was applied to the temperature inputs, and a +/- 5% was applied to the RH inputs. The graph shows that even when applying a fixed change in temperature or RH, the sensitivity of Popmite varies, depending on the base-case conditions to which such changes are applied.

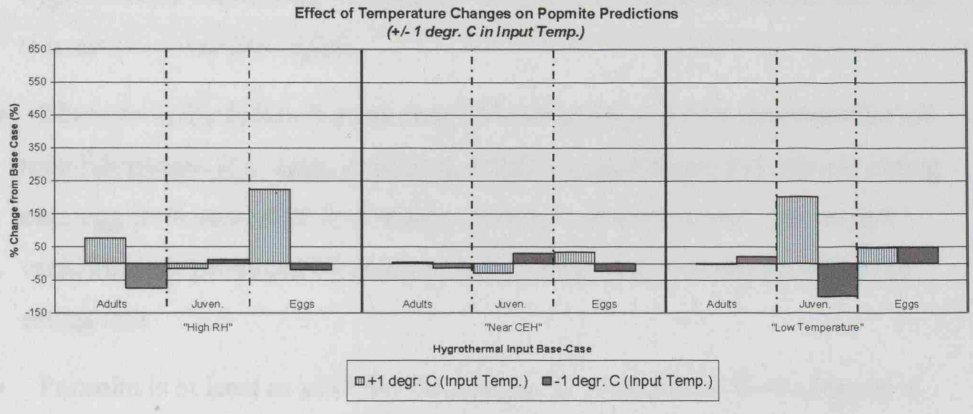


Fig 9.4.6

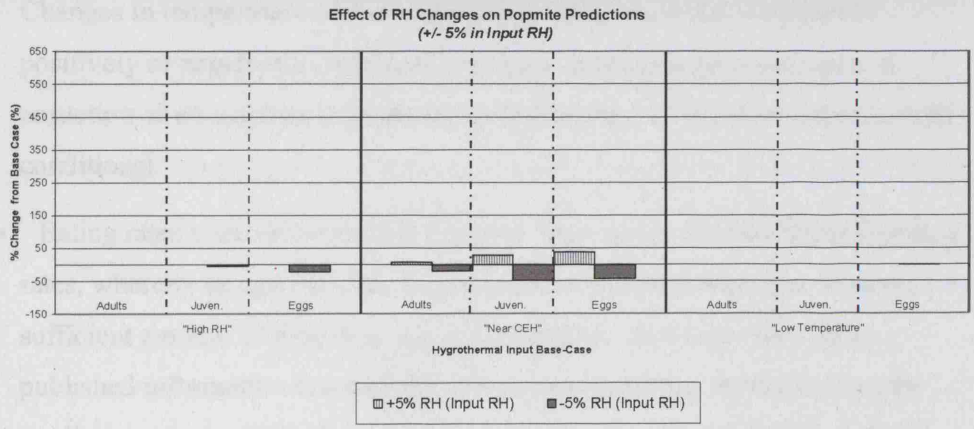


Fig 9.4.7

Fig 9.4.6-7 Effect of fixed changes in temperature or RH on Popmite predictions. Starting population: 20 adults, spread of all ages.

So far, the sensitivity analysis results for the Popmite 7d model have been described, which resulted from utilising an initial population of 20 adults, with a spread of all ages. The sensitivity of Popmite to the starting population has also been investigated (see Appendix A.9, where different initial populations were investigated: i.e. 20 juveniles; 20 eggs; or 20 *fresh* adults). However, no significant differences were observed (from what was described so far) with changes in the life stage or the age of the starting population.

This section described a sensitivity analysis for the Popmite 7d model, which was found to be a complex model, with strong threshold effects. In summary:

- The impact of changes in input parameters is dependent on the input hygrothermal conditions, and it also varies in relation to the output life stage (i.e. adults, juveniles, eggs).
- Changes in the following parameters have an impact on predictions for *all* mite life phases (i.e. eggs, juveniles, adults): temperature, RH, female eating rate, egg food cost, max food intake, and base mortality rate. The largest variations are produced by changes in hygrothermal conditions and by the eating rate.
- Popmite is at least as sensitive to changes in temperature as to changes in RH, and in some cases changes in temperature have the largest impact. Changes in temperature and changes in predictions are not consistently positively or negatively correlated (e.g. greater temperatures can cause a reduction *or* an increase in predictions, depending on the initial hygrothermal conditions).
- Eating rates were introduced in Popmite 7d as a way of controlling breeding rates, whereby an egg can only be produced when the female has consumed a sufficient amount of food (and enough moisture). However, very little published information is available on these mechanisms, to which Popmite predictions are particularly sensitive. Consequently, it is recommended that experiments are carried out on eating/egg-laying rates, in order to fine-tune the Popmite predictions.

9.5 Summary discussion

This chapter discussed the sensitivity analysis carried out for three of the four hygrothermal and population models to their input variables. The sensitivity analysis of the BED model was also briefly discussed, based on the results from Pretlove *et al.* (2005). The main aims of the sensitivity analysis were to identify those input variables which need to be known with greater accuracy, and which ultimately have the greatest impact on mite growth/decline predictions.

In summary, the results suggest that all 4 models are sensitive to input hygrothermal conditions. In particular:

- The mattress hygrothermal models are mostly sensitive to room conditions, followed by boundary conditions (particularly temperature), and - to a lesser extent - to the length of time the bed is occupied.
- The population models are sensitive to input hygrothermal conditions. However, the models' sensitivity also depends on the hygrothermal conditions to which such changes are applied (i.e. base-case conditions). For example, if the hygrothermal conditions are extremely unfavourable for mite growth, a 10% change in hygrothermal conditions does not change the predictions. In both models threshold effects can be observed, often in unpredictable ways. Therefore, it is not possible to identify a *typical* change in prediction, due to a specific change in input hygrothermal conditions.
- In both population models, changes in RH and predictions are positively correlated (i.e. an increase in RH leads to greater predicted mites). However, an increase in temperature might lead to a decrease or increase in predictions, depending on the hygrothermal conditions to which such changes are applied (base-case).
- In most cases, the MPI model is more sensitive to changes in RH, than to changes in temperature. However, depending on the base-case hygrothermal conditions and on the size of the change, changes in temperature can be important as well - particularly at low base-case RHs. The Popmite model is at least as sensitive to changes in temperature as to changes in RH, and in some cases changes in temperature have the largest impact.
- The largest variations in Popmite predictions are not only produced by changes in hygrothermal conditions, but also by changes in the eating rate. The sensitivity of Popmite also changes in relation to different output types (i.e. adults, juveniles and eggs).

These results suggest that it is particularly important to determine as accurately as possible the input hygrothermal conditions for all the models considered in this thesis. Accuracy in temperature inputs should not be ignored, since these can be as important as RH inputs – both for the population models and for the input boundary conditions in the mattress models. The threshold effects and the sensitivity to both temperature and RH observed in both population models

might partly explain why some intervention studies on the psychrometric control of house dust mites have been unsuccessful.

Input boundary conditions play a role in the mattress models' predictions, particularly for Lectus. The comparison with the fieldwork results for the boundary conditions in Lectus revealed that there is a certain degree of variability in such conditions both within and between individuals (see Chapter 5). Therefore, this variability should be taken into account in any scenarios modelling.

Since Popmite is also very sensitive to eating rates, it is recommended that experiments are carried out on eating (and egg-laying) rates, where little published information is currently available.

It should also be highlighted that the size of the starting population and the time (when and for how long the mites are exposed to specific hygrothermal conditions) are also going to determine whether a population survives unfavourable hygrothermal conditions.

Chapter 9: References

Biddulph P, Crowther D, Leung B, Wilkinson T, Hart B, Oreszczyn T, Pretlove S, Ridley I, Ucci M, (2007), Predicting the population dynamics of the house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle, *Experimental and Applied Acarology*, 41(1-2): 61-86.

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

Oreszczyn T and Pretlove S E C, (1999), Condensation Targeter II: Modelling surface relative humidity to predict mould growth in dwellings, *Building Services Engineering Research and Technology*, 20 (3) 143 – 153.

Pretlove SEC, Oreszczyn T, Ridley I, Wilkinson T, Crowther D, (2005), A steady-state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements, *Building Services Engineering Research and Technology*, 26(4): 301-314.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (submitted), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air.*

CHAPTER 10:

COMPARISON OF SIMPLE VERSUS COMPLEX MODELS

CHAPTER 10: COMPARISON OF SIMPLE VERSUS COMPLEX MODELS

10.1 Introduction

This thesis aims to test the hypothesis that a combined HDM population-hygrothermal model for beds can adequately predict field data and that the model can be a valuable tool for scenario modelling and intervention studies focused on the psychrometric control of house dust mites. In particular, the thesis tests 2 sets of combined hygrothermal population models: the "simple" BED-MPI steady-state models, and the "complex" Lectus-Popmite transient models. More specifically, the simple population model MPI is a steady-state model, while the complex population Popmite is transient. The simple bed model BED is a steady-state one-dimensional model, whilst the complex Lectus model is a transient 3-dimensional model. The previous chapters discussed model validation against fieldwork data and a sensitivity analysis for the models. This chapter discusses the main differences in predictions between the simple and the complex models, in comparison with fieldwork data. The comparison between the two sets of model aims to:

1. Assess whether the additional complexities of the complex models (Lectus/Popmite) result in predictions which match the fieldwork data more closely than the simple models (BED/MPI), or whether in most circumstances the simple models could provide sufficiently satisfactory predictions for scenarios modelling and/or intervention studies.
2. The simple and the complex models were developed independently and with different approaches. If their predictions for the same hygrothermal conditions agree, this represents a confirmation of their validity.

Although Popmite was developed for transient hourly hygrothermal input conditions, the model could also theoretically be used with constant hygrothermal inputs, repeated over a certain time-period. Section 10.2 compares the Popmite predictions obtained by using *constant* hygrothermal conditions against the predictions obtained by using *transient* hygrothermal conditions, and against the fieldwork results ("mite cages").

Section 10.3 compares the predictions of the simple versus the complex population models (MPI and Popmite respectively), while Section 10.4 compares the predictions of the simple versus the complex hygrothermal bed models (BED and Lectus respectively).

The chapter ends with a summary discussion in section 10.5.

10.2 Popmite: transient versus constant input conditions

Popmite is a model which predicts the effects of hygrothermal conditions on each life cycle phase of a house dust mite (i.e. eggs, juvenile, adults). The model can be tailored to any particular species of domestic mite. In this thesis, the *Popmite Version 7d* is utilised, which is a further development of the model described by Biddulph *et al.* (2007). Popmite 7d predicts the impact of *transient* hygrothermal conditions on *wild Dermatophagoides pteronyssinus* (DP) mites, feeding on a natural diet (Hart *et al.*, 2007). Although the population model Popmite 7d was developed for *transient* hourly hygrothermal input conditions, the model could also theoretically be used with *constant* hygrothermal conditions, repeated over a certain time-period. For example, the outputs of the BED model (monthly average temperature and RH in the bed core) could be utilised in Popmite by repeating them as constant conditions over one month, in order to assess the likely impact of these hygrothermal predictions on mite growth. This section compares the Popmite predictions obtained by using *constant* hygrothermal conditions against the predictions obtained by using *transient* hygrothermal conditions, and against the fieldwork results ("mite cages"). If the predictions obtained by using the two methods were similar (at least in certain circumstances), than it could be concluded that average conditions are sufficient for population modelling. Therefore, it would not be necessary to obtain transient hygrothermal data (monitored or predicted) for room conditions, which would simplify the research.

Figure 10.2.1 shows the comparison between the mite "cages" results[1] and the predictions of Popmite 7d corresponding to *constant* input hygrothermal conditions. Since each "mite bag" was exposed to monitored transient hygrothermal conditions over a period of n hours (n ~ 1008), for each mite cage

[1] See Chapter 4 for a description of fieldwork.

the constant hygrothermal conditions were obtained by repeating for n hours the *average* temperature and RH to which the cages were exposed[2]. Figure 10.2.1 can be compared with the results obtained by using *transient* input hygrothermal conditions (Figure 10.2.2).

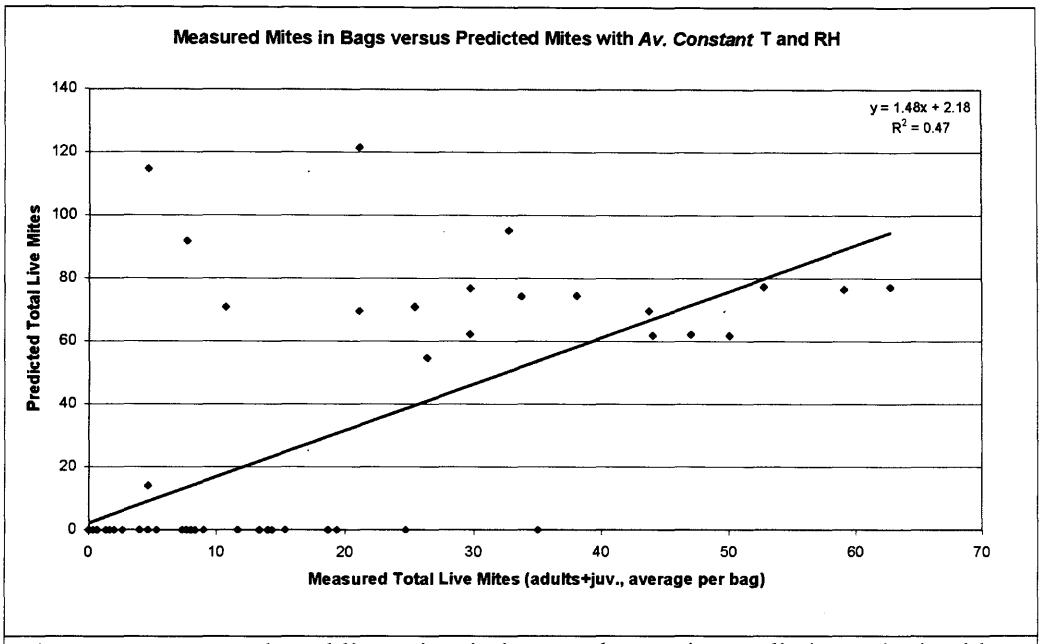


Fig 10.2.1 Measured total live mites in bags and Popmite predictions obtained by using constant *average* hygrothermal input conditions.

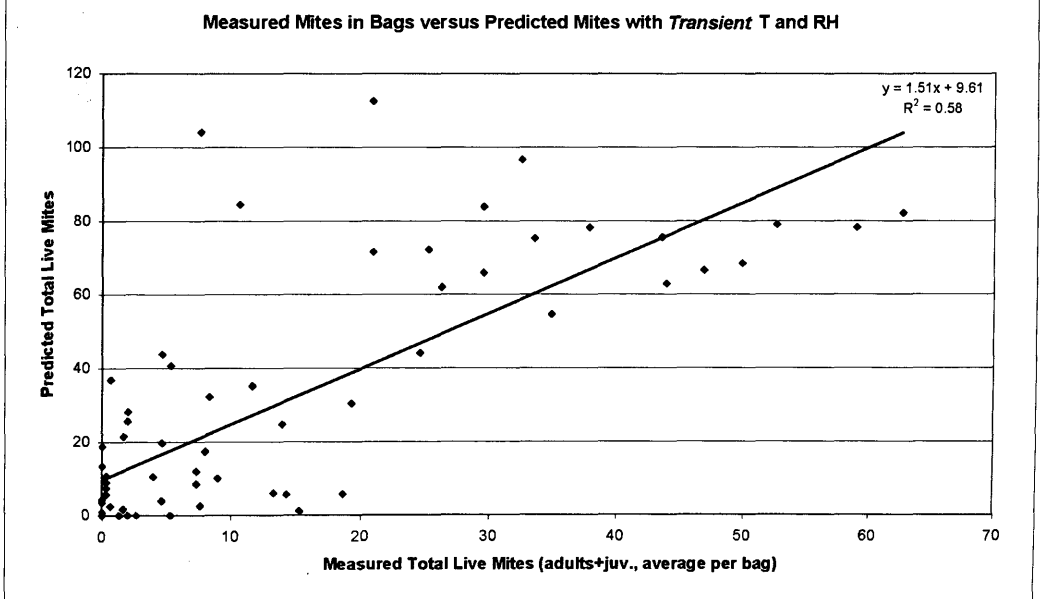


Fig 10.2.2 Measured total live mites in bags and Popmite predictions obtained by using *transient* hygrothermal input conditions.

[2] In this section the mite cages results from the Series 1 study (see Chapter 4) are excluded from the data analysis, since in Series 1 laboratory-reared mites were utilised in the mite bags, as opposed to wild mites utilised in the Series 2 and 3 studies.

The graphs show that transient inputs lead to a better agreement with fieldwork results. This is perhaps unsurprising, since the mite bags were exposed to highly transient conditions. In both cases (transient or constant inputs), the model over-predicts by a factor of approximately 1.5. The main difference between the 2 cases is that for low measured mite numbers (<20), the use of constant hygrothermal inputs is more likely to lead to nil predictions, than the case when transient hygrothermal inputs are utilised. This is confirmed by Figure 10.2.3, which also shows that predictions obtained from constant inputs and predictions obtained from transient inputs are in good agreement (R-squared value 0.81), particularly for those cases where predicted mite numbers are higher - although constant inputs lead to marginally lower predictions than transient inputs.

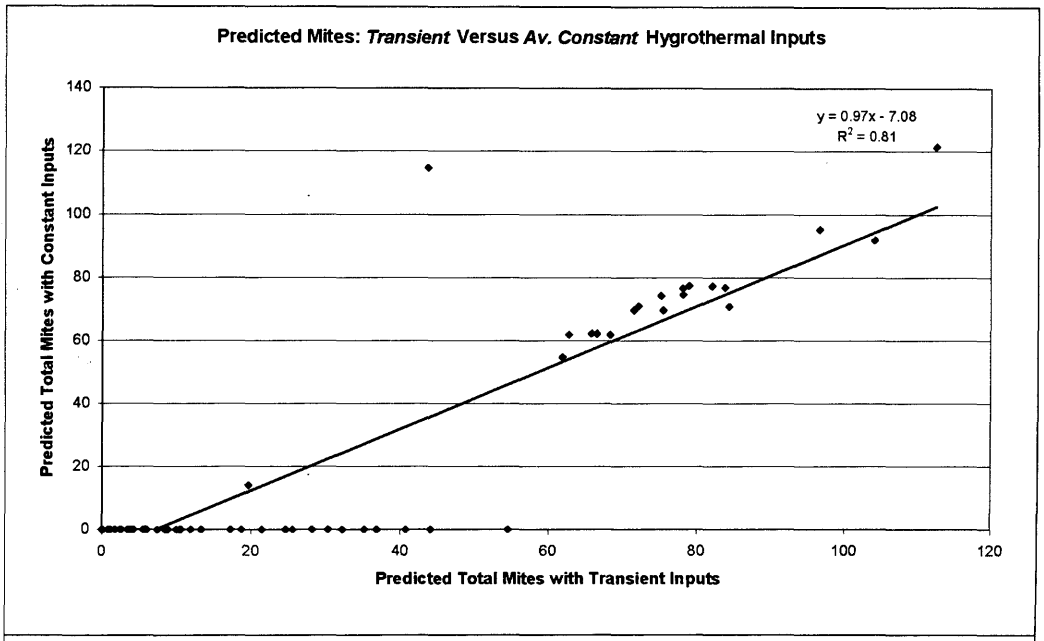


Fig 10.2.3 Popmite predictions using *transient* hygrothermal inputs versus predictions using *constant* hygrothermal inputs.

Figure 10.2.4 and 10.2.5 show the average RH (measured in the mite bags) versus the difference between predicted and measured mites. The graphs suggest that the main difference between using constant or transient inputs occurs when the average RH is in mid-range (45-60%), where constant inputs result in under-predictions, while transient inputs result in over-predictions. This is most probably because those mid-range conditions are very close to the Critical Equilibrium

Humidity (CEH) in the mite cages, which was on average 56% (standard deviation: 0.6%).

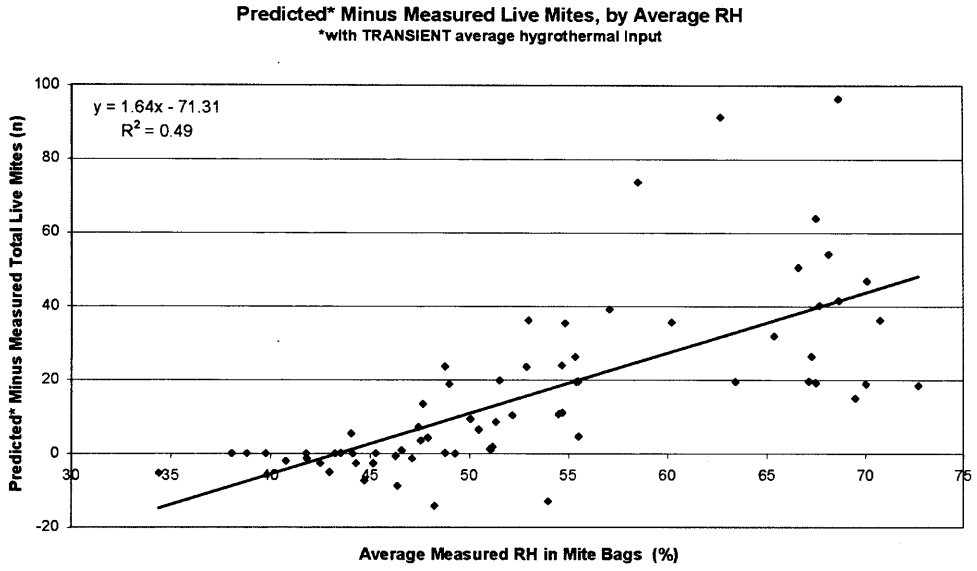


Fig 10.2.4 Predicted* minus measured live mites by average RH (*using *transient* hygrothermal inputs).

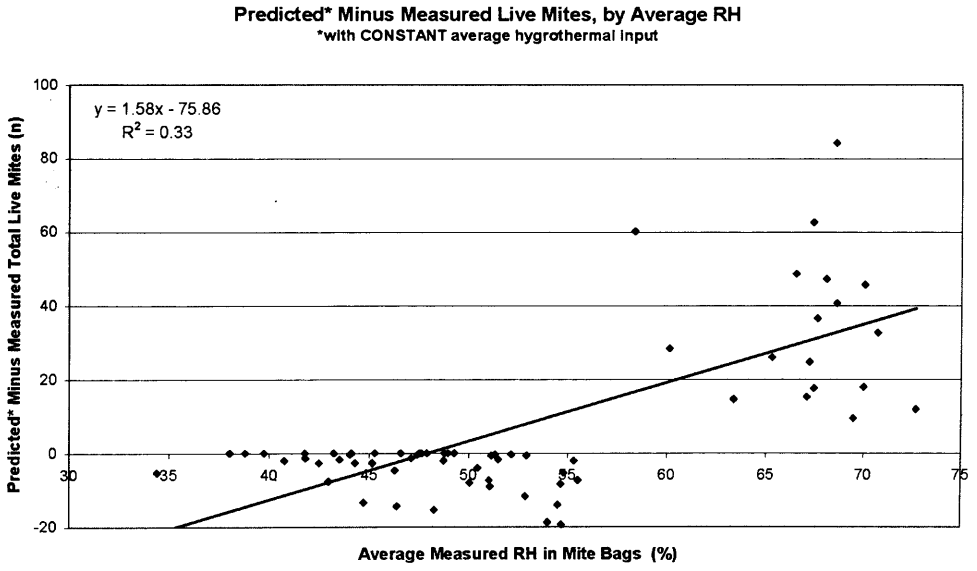


Fig 10.2.5 Predicted* minus measured live mites by average RH (*using *constant* hygrothermal inputs).

The results from Figure 10.2.4-5 are confirmed by Figures 10.2.6 and 10.2.7, which show the percentage of time the RH in the mite bags was greater than the

Critical Equilibrium Humidity (CEH)[3], compared with the difference between predicted and measured live mites. When the percentage of time the RH>CEH is between 10-40%, the predictions obtained by using transient inputs are higher than measured values, while the predictions obtained by using constant inputs are lower than measured values.

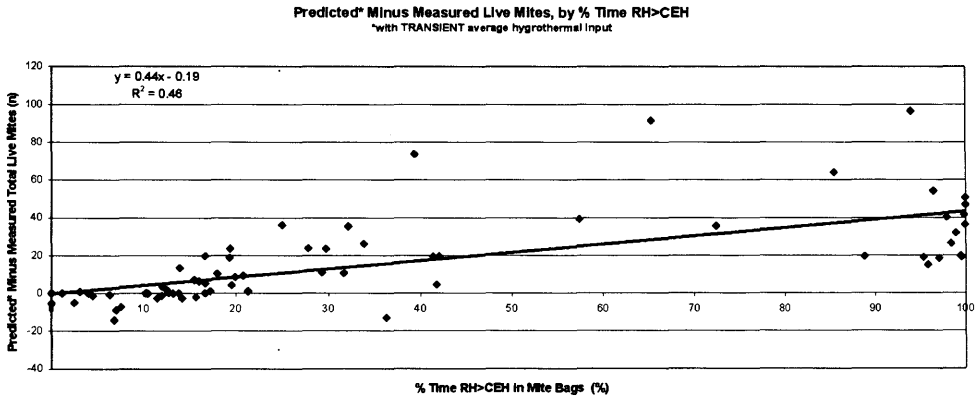


Fig 10.2.6 Predicted* minus measured live mites by percentage of time RH>CEH (*using *transient* hygrothermal inputs).

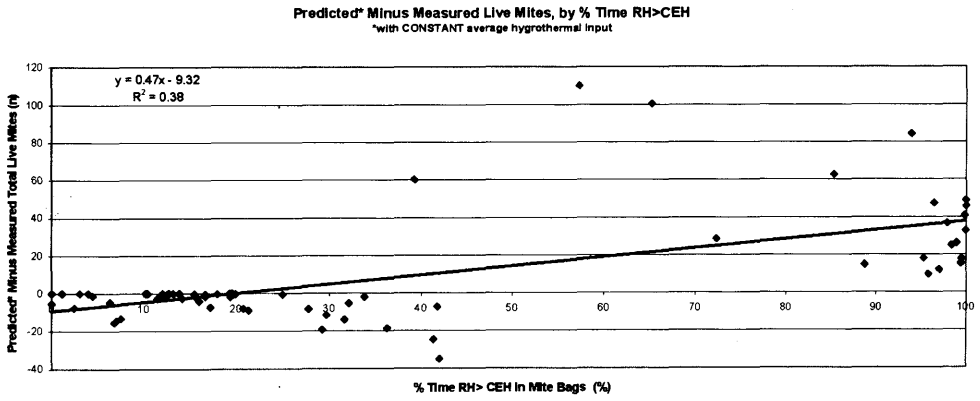


Fig 10.2.7 Predicted* minus measured live mites by percentage of time RH>CEH (*using *constant* hygrothermal inputs).

Temperature did not have a significant impact on the difference between predicted and measured results, neither for the transient nor for the constant inputs. However, constant inputs resulted in predictions which differed the most from measured values when the average temperature in the mite bags was between 18- and 23 °C.

[3] CEH=54.5-0.005*temp+[525.6/(temp-39.3)^2], only valid if temp. < 37 °C. This is the CEH utilised in Popmite (see Chapter 7).

This section compared the Popmite predictions obtained by using *constant* hygrothermal conditions against the predictions obtained by using *transient* hygrothermal conditions, and against the fieldwork results ("mite cages"). The comparison aimed to assess whether the complexity of transient modelling provides better accuracy in predictions. The results indicate that:

- Overall, Popmite predictions obtained by utilising *transient* hygrothermal inputs fits the mite bags results better (R-squared value: 0.58) than the predictions obtained by utilising *constant* hygrothermal inputs (R-squared value: 0.47).
- Transient and constant predictions are on the whole in agreement with each other and with measured data, if the average RH is very low (<40%) and well below CEH (% time RH>CEH: smaller than 10%).
- If the average RH is fairly high (>60-65%) and well above CEH (% time RH>CEH: greater than 60-70%), both transient and constant predictions are quite similar, predicting higher mite numbers than measured value, with a fairly low correlation with measured mites.
- If however the average RH is between 45-60% - a condition typical in many UK dwelling -and/or the percentage of time the RH is above CEH is between 10-40%, then transient inputs over-predict measured results, while constant inputs under-predict them. The transient inputs lead to slightly better agreement with measured values – although with over-predictions.

These results suggest that if mite populations are exposed to transient conditions, in general it is more advisable to utilise Popmite with transient hygrothermal inputs, particularly for hygrothermal conditions which are rather close to the Critical Equilibrium Humidity.

The next section compares the Popmite predictions with the MPI predictions.

10.3 Popmite and MPI: comparison

This section discusses the comparison between the two population models (MPI and Popmite), against the fieldwork results (mite bags). The comparison between measurements and predictions has already been discussed separately for each

model, in Chapter 7 (Popmite) and 8 (MPI). From those two Chapters it emerged that MPI predictions fit the field measurements less accurately than Popmite predictions (MPI R-squared value: 0.49; Popmite R-squared value: 0.58). This was somewhat expected, since the mite bags were kept under transient conditions. Furthermore, the experiments which formed the basis of the MPI model included a larger population of laboratory-reared mites, as opposed to a smaller population of *wild* mites on a "natural" diet (Series 2 and 3 mite bags).

Although the MPI model predicts the mite bags results less accurately than the Popmite model, MPI predictions fit the results a little more accurately than the Popmite model when *constant* input hygrothermal conditions are utilised (see section 8.2: R-squared value 0.47). In all three cases, the model tends to over-predict by a factor of approximately 1.5.

It is possible to directly compare the MPI predictions with the Popmite ones, by calculating the MPI factor for the Popmite results. This is simply the Popmite prediction of mites after n hours, divided by the starting population (e.g. 20 mites for Series 2 and 3 mite bags).

Figure 10.3.1-2 shows the MPI predictions, compared with the Popmite predictions obtained from constant inputs (Figure 10.3.1) and from transient inputs (Figure 10.3.2). The graphs show that there is reasonable agreement between the 2 models, with a marginally better agreement when transient inputs are used in Popmite, as opposed to constant ones.

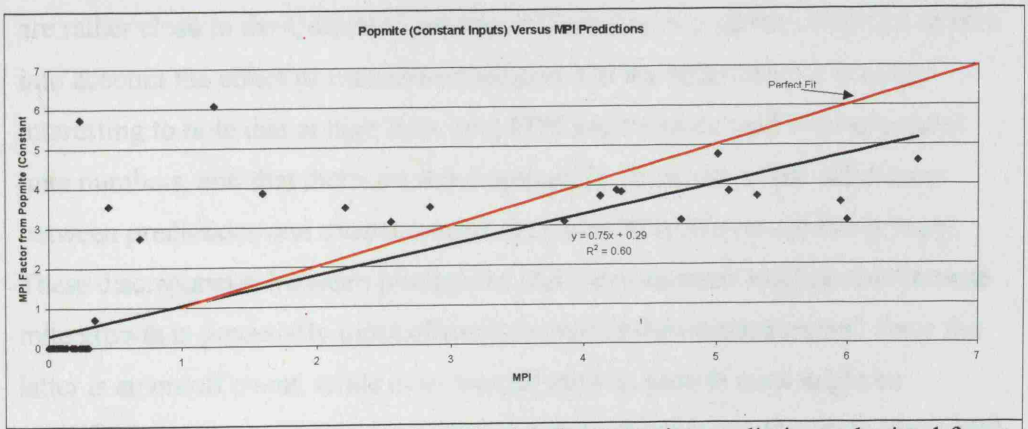


Fig 10.3.1 MPI predictions, compared with the Popmite predictions obtained from constant inputs.

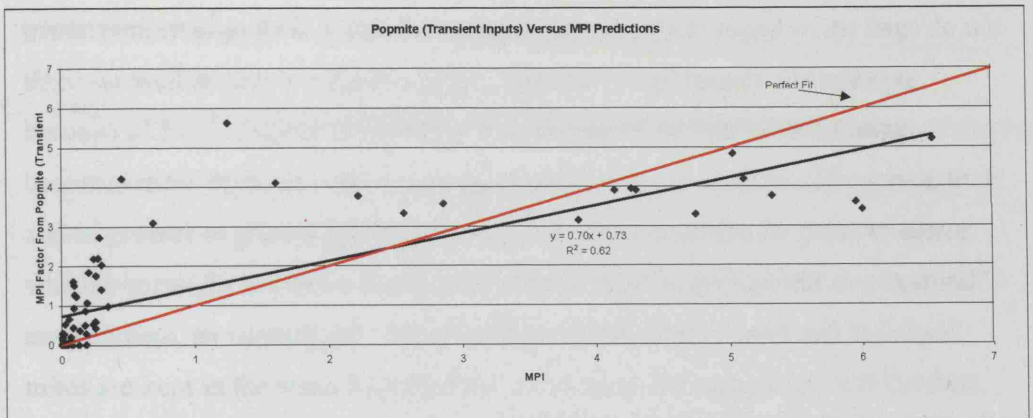


Fig 10.3.2 MPI predictions, compared with the Popmite predictions obtained from transient inputs.

Figures 10.3.3-5 show the difference between predictions and measurements, by the average RH measured in the mite bags. The graphs show that in all 3 cases (Popmite transient, Popmite constant and MPI) the predictions are fairly similar to each other at low average RHs and at high average RHs. In particular, at low average RHs (<40%) the models predict low mite numbers, mostly in agreement with the mite bags measurements - although with a tendency for under-predictions. At high average RHs (>60-70%), the models predict higher than measured mite numbers. At mid-range average RHs (45-60%) the Popmite predictions from *transient* inputs over-predict but agree with measured results a little better than when *constant* inputs are utilised in Popmite, or when MPI is used. This is most probably because when the measured hygrothermal conditions are rather close to the Critical Equilibrium Humidity, it is rather important to take into account the effect of transient conditions. On the other hand, it is quite interesting to note that at high RHs both MPI and Popmite tend to over-predict mite numbers, and that there are some noticeable variations in the differences between predictions and measurements at similar RHs (if average RH is high). These discrepancies between predictions and measurements might occur because mite growth is potentially more difficult to predict than death/survival, since the latter is an on/off event, while even a small error in growth rates might be amplified over time, leading for example to over-predictions. However, since MPI and Popmite were developed independently and with 2 rather different approaches, it is quite a coincidence that both models should over-estimate mite growth in a similar manner. An alternative explanation for the models' over-

predictions at high RHs is that for some reason the mites caged in the bags do not thrive as well as they would in a more "natural" environment, for example because of food or space constraints. It is also possible that mite counting becomes more difficult with a high number of mites, leading to under-predictions and in general to greater differences with *real* mite numbers. In order to assess whether mites do not thrive in the mite bags as well as they would in a "natural" environment, an experiment should be carried out, where caged and un-caged mites are kept in the same hygrothermal conditions for some time, and the final mite numbers are counted in each case. A calibration process for the acarologist performing the mite counting should also be implemented.

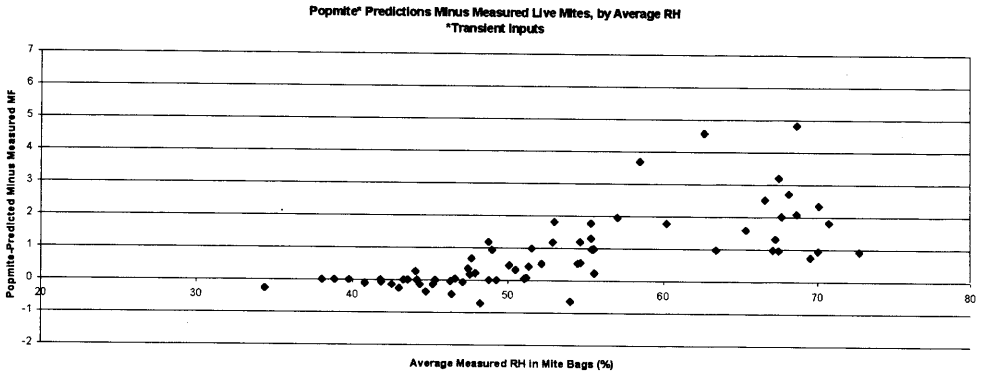


Fig 10.3.3 Average RH measured in the mite bags, and difference between predicted and measured results (expressed in MPI terms), for Popmite with transient inputs.

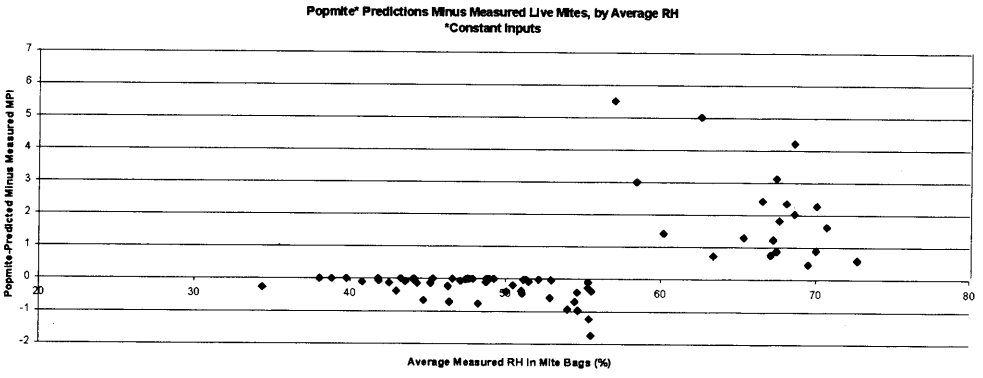


Fig 10.3.4 Average RH measured in the mite bags, and difference between predicted and measured results (expressed in MPI terms), for Popmite with constant inputs.

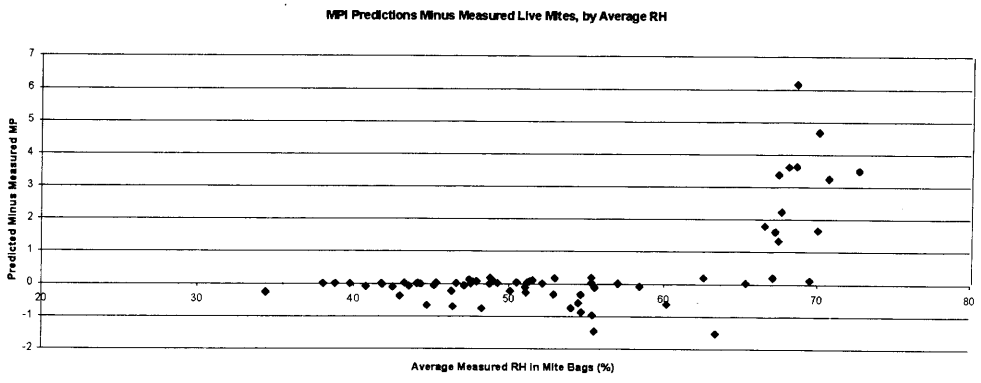


Fig 10.3.5 Average RH measured in the mite bags, and difference between predicted and measured results(expressed in MPI terms), for MPI.

This section compared the MPI and the Popmite models. The results show that Popmite predictions from transient hygrothermal inputs have a better agreement with mite bag results (R-squared value: 0.58) than MPI's predictions (R-squared value: 0.49). This is somewhat expected, since MPI was developed for steady-state conditions, and for a larger starting population. On the other hand, MPI predictions are marginally better than Popmite predictions from constant inputs (R-squared value: 0.47). However, these conclusions might differ for larger starting populations and/or steady-state hygrothermal conditions, since MPI was developed for steady-state conditions with a starting population 50 times bigger than the starting population in the mite bags.

The main difference in predictions between the steady-state and the transient models occurs when average RHs are close to the Critical Equilibrium Humidity – in which case Popmite (with transient inputs) performs better than the steady-state models.

Some striking similarities occur between Popmite and MPI predictions. For example, both Popmite and MPI over-predict mite bags measurements, particularly at high average RHs. Also, at high RHs there are some noticeable variations in the differences between predictions and measurements at similar RHs, for both models. These similarities are reassuring, since the two models were developed independently using two fundamentally different approaches (theoretical/empirical). However, at present it is unclear what might be causing these over-predictions. One possibility is that in favourable conditions the caged mites do not thrive in the bags as well as they would in a "un-caged" environment. Mite counting might also results in a systematic error at high mite numbers because mite counting may be difficult under those conditions. In order to determine whether this might be the case, further experiments should be carried on mite bags, with a calibration protocol for mite counting. Another explanation for the population models' over-predictions might be that the models do not explicitly include restrictions for food or space availability, which did occur in the mite bags. Competition with moulds might also occur at high RHs, which could affect mite growth in the mite bags. However, the impact of mould is not explicitly considered in either population models.

Since the comparison with fieldwork results suggest that the population models might over-predict mite growth, the models can be used in scenarios modelling for comparison purposes, in order to assess which scenario lead to greater/lower mite numbers. However, at present the models might underestimate the number of techniques capable of *eradicating* mites altogether.

So far the two population models have been compared. The next section discusses the comparison between the two hygrothermal bed models.

10.4 BED and Lectus: comparison

In this section the predictions from the hygrothermal bed models BED and Lectus are compared with each other and against fieldwork data.

The BED model (Pretlove *et al.*, 2005) is a steady-state one-dimensional hygrothermal model, which predicts the average monthly temperature and RH within the bed core (the occupied space between mattress and covering), given the average monthly temperature and RH of the bedroom. The Lectus model (Ridley *et al.*, submitted), is a transient 3-dimensional heat and water vapour transfer model. Lectus predicts hourly temperature and RH in a bed (3-dimensional array of cells), given hourly room conditions and the mattress properties. In Lectus the mattress boundary conditions are the same as the room when the bed in not occupied. When the bed is occupied, an empirical set of boundary conditions are utilised on the top surface of the mattress[4] (see Chapter 3). Since the BED model predicts bed conditions corresponding to the bed core (i.e. top mattress surface, under chest area), the BED predictions should be compared with the Lectus predictions for the same mattress area. These predictions in Lectus are independent from mattress properties, since they are only driven by the empirical boundary conditions. In particular, in Lectus it is assumed that under the chest area the boundary conditions are 34 °C and 1000 Pa (vapour pressure excess) when the bed is occupied, whilst they are the same as the room when the bed is unoccupied.

[4] In Lectus the bottom and the sides of the mattress have the same conditions as the room, at all times.

In order to compare the BED and Lectus predictions with each other and against field data, the monitored room conditions from the Series 1 and 2 study were considered. The results from Series 3 were excluded, for 2 main reasons: a) Series 3 monitored children, who might be governed by different thermo-regulatory processes than adults, whilst BED and Lectus were developed for adults; b) an additional padded cover was used in Series 3 to cover the bed sensors, which represents a confounding element when analysing the monitored bed results.

For each monitored bed from Series 1 and 2 (12 beds overall), the surface conditions corresponding to the chest area predicted by Lectus were calculated by considering the monitored room conditions and assuming that the bed was occupied for a number of hours per night, which had been reported by the bed occupant as the average number of sleeping hours per day. In this way, predicted hourly values were obtained for the bed core area, which were the same as the monitored room conditions, except when the bed was occupied (e.g. 8 hours per night), when the conditions were 34 °C and 1000 Pa (vapour pressure excess)[5]. For each bed, the average temperature and RH were then calculated from these values, in order to enable a comparison with the BED predictions. The main uncertainties for the Lectus bed core predictions are linked to the input room conditions (i.e. sensors' accuracy).

The BED model predictions were calculated following the same procedure described in Chapter 6. This involved considering the measured room conditions, the measured mattress thickness and a number of hours per day which the occupant reported as the average daily sleeping hours. Although the BED model requires other input variables, these were not measured during the fieldwork study and therefore the default values were not modified. The uncertainties in predictions related to uncertainties in input parameters were also calculated, according to the procedure described in Chapter 6.

Figure 10.4.1 and 10.4.2 compare the two model's predictions with the measurement results, respectively for the temperature and the RH. For each BED data point in the graphs, the vertical error bars correspond to the uncertainties due to input variables. The vertical error bars for the Lectus data points correspond to the uncertainties in predictions due to *measurement* of the input room conditions

[5] In Lectus when the bed is vacated, the mattress conditions go back to room conditions gradually.

(i.e. loggers accuracy). The horizontal error bars represent the error measurement, corresponding to the loggers' accuracy.

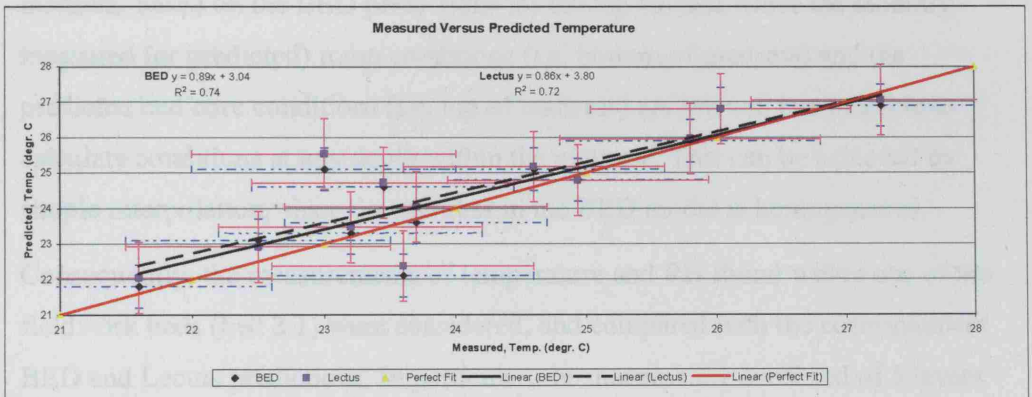


Fig 10.4.1 BED and Lectus predictions: measured versus predicted temperature.

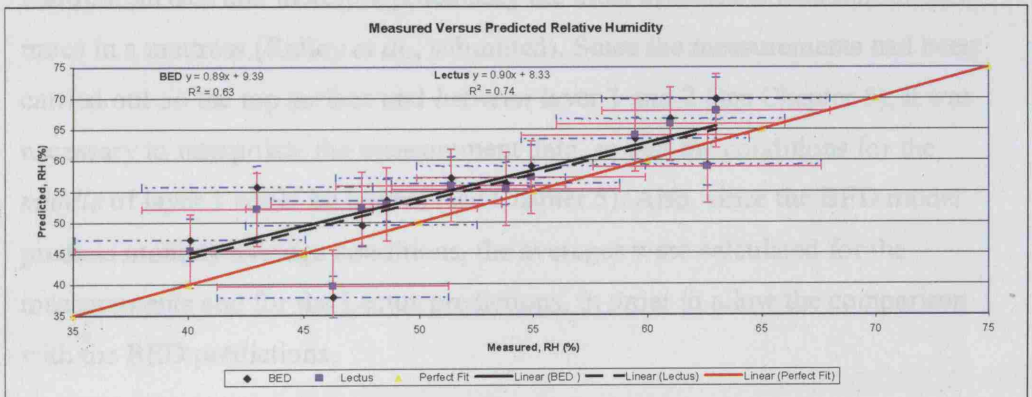


Fig 10.4.2 BED and Lectus predictions: measured versus predicted RH.

Figure 10.4.1 and 10.4.2 show that there is a good agreement between the 2 models and with measured results. The BED model predicts the temperature slightly better than the Lectus model. However, Lectus predictions match more closely the measured RH results, than the BED predictions.

As already mentioned in Chapter 6, the conditions in the bed core are not necessarily the most favourable to house dust mites. In fact, the MPI for the bed core is lower than for room conditions. Detailed measurements showed that other positions within the mattress may be more favourable for mite colonisation - provided physical access is achievable at these other locations and food is accessible (Pretlove *et al.*, 2005). Ridley *et al.* (submitted) found that conditions

might be most favourable to HDM growth at 1-2 cm below the mattress top surface. Consequently, it may be of interest to calculate conditions *within* the mattress, based on the BED predictions for the top surface. Once the monthly measured (or predicted) room conditions (i.e. bottom of mattress) and the predicted bed core conditions (i.e. top of mattress) are known, it is possible to calculate conditions at any depth within the mattress. This can be achieved by simple interpolation, since the mattress in the BED model is homogeneous.

Consequently, the measurements of temperature and RH found within one of the fieldwork beds (bed 2.1) were considered, and compared with the correspondent BED and Lectus predictions. In particular, the mattress 2.1 consisted of 5 layers (see Chapter 5). It was decided to consider the predictions from Lectus and BED for the *middle*[6] of layer 1 (under the chest area), which is at 1.25 cm from the top mattress surface and therefore potentially the most favourable location for dust mites in a mattress (Ridley *et al.*, submitted). Since the measurements had been carried out on the top surface and *between* layer 1 and 2 (see Chapter 5), it was necessary to interpolate the measurement data, so that the conditions for the *middle* of layer 1 could be known (see Chapter 5). Also, since the BED model predicts monthly average conditions, the averages were calculated for the measurements and for the Lectus predictions, in order to allow the comparison with the BED predictions.

Figure 10.4.3 (temperature) and 10.4.4 (RH) show the comparison between measurements and predictions for the mattress surface and for layer 1. The error bars due to input uncertainties and to measurement uncertainties - which were calculated as described in Chapter 5 and 6 - are also plotted. The graphs show that once these uncertainties are taken into account, the two models both predict measurement results satisfactorily. It should be mentioned that BED predictions for layer 1 were initially calculated by interpolation. However, although in the BED model the mattress is modelled as a homogenous layer, the real mattress consisted of 5 layers. Therefore, it was found that the BED predictions for layer 1 matched measurement results better, if the Glaser Method was utilised (BSI, 2002) - rather than simple interpolation.

[6] Lectus predicts conditions at the *middle* of each layer

It should be pointed out that although Lectus is a 3-dimensional model while BED is a 1-dimensional model, the effect of 3-dimensional heat and moisture transfer is less marked in the centre of the mattress – where the BED model is predicting. Since no significant differences were found (once uncertainties were considered) between the BED and the Lectus predictions for the point 1.25 cm below the top surface (chest area), it appears that the effect of 3-dimensonal heat and moisture transfer is smaller than the uncertainties, at the point of interest.

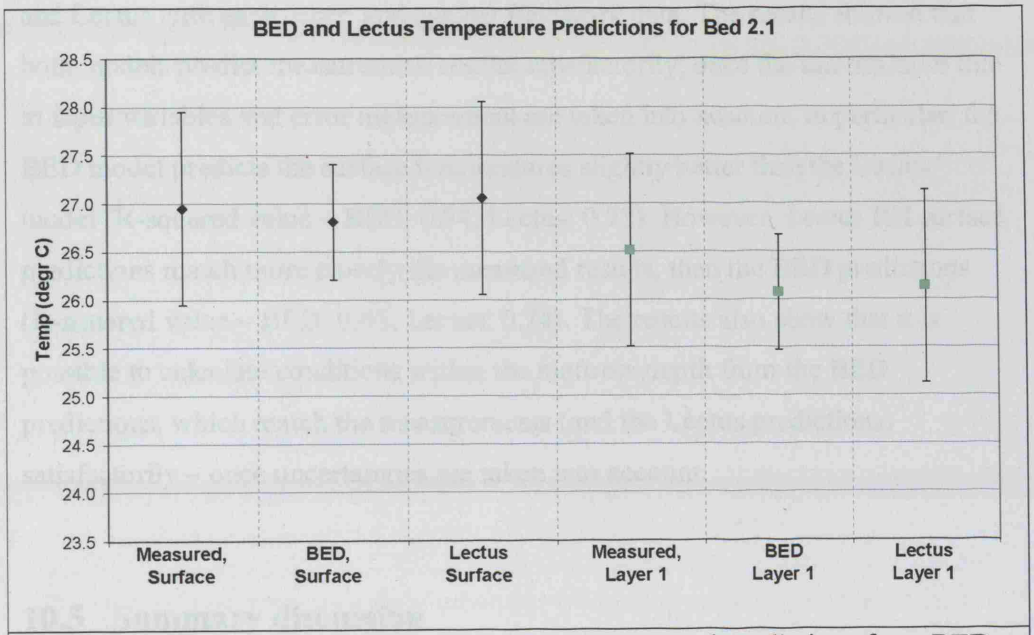


Fig 10.4.3 Comparison of temperature measurements and predictions from BED and Lectus, for Bed 2.1.

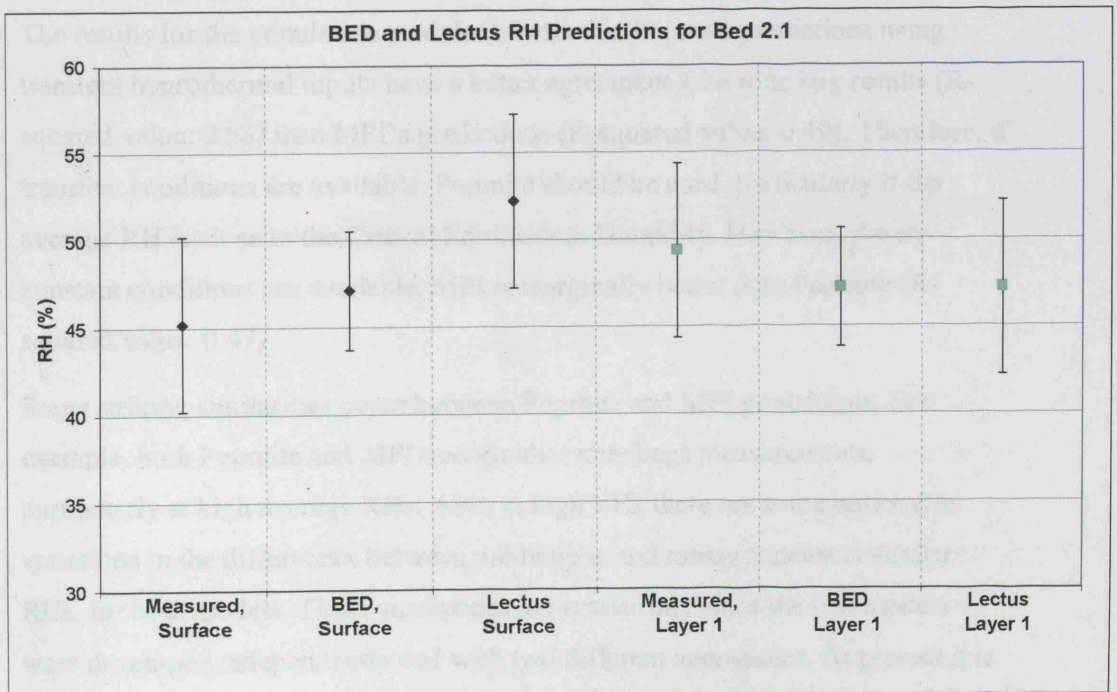


Fig 10.4.4 Comparison of RH measurements and predictions from BED and Lectus, for Bed 2.1.

This section compared the predictions from the hygrothermal bed models BED and Lectus with each other, and against fieldwork data. The results showed that both models predict measurement results satisfactorily, once the uncertainties due to input variables and error measurement are taken into account. In particular, the BED model predicts the surface temperatures slightly better than the Lectus model (R-squared value – BED: 0.74, Lectus: 0.72). However, Lectus RH surface predictions match more closely the measured results, than the BED predictions (R-squared value – BED: 0.63, Lectus: 0.74). The results also show that it is possible to calculate conditions within the mattress depth from the BED predictions, which match the measurements (and the Lectus predictions) satisfactorily – once uncertainties are taken into account.

10.5 Summary discussion

This chapter discussed the main differences in predictions between the simple and the complex hygrothermal population models.

The results for the population models showed that Popmite predictions using transient hygrothermal inputs have a better agreement with mite bag results (R-squared value: 0.58) than MPI's predictions (R-squared value: 0.49). Therefore, if transient conditions are available, Popmite should be used, particularly if the average RH is close to the Critical Equilibrium Humidity. However, if only constant conditions are available, MPI is marginally better than Popmite (R-squared value: 0.47).

Some striking similarities occur between Popmite and MPI predictions. For example, both Popmite and MPI over-predict mite bags measurements, particularly at high average RHs. Also, at high RHs there are some noticeable variations in the differences between predictions and measurements at similar RHs, for both models. These similarities are reassuring, since the two models were developed independently and with two different approaches. At present it is unclear what might be causing these over-predictions. One possibility is that the caged mites do not thrive in the bags as well as they would in a "natural" environment. Mite counting might also be more difficult with high mite numbers, which could lead to under-estimation and/or inaccurate results. In order to

determine whether this might be the case, further experiments should be carried out, where two mite populations with the same size are held in identical hygrothermal conditions, but one population is kept in mite bag(s), and another population is held in a more "natural" environment. Careful thought should be given on the amount of food provided to each population. A calibration protocol for mite counting should also be adopted.

Since the comparison with the mite bags results suggest some agreement with field data – although possibly with a tendency towards over-prediction - the models can be used in scenarios modelling for comparison purposes, in order to assess which scenario might lead to greater/lower mite numbers. However, at present the models might underestimate the number of techniques which can effectively *eradicate* mites altogether.

This chapter also compared the predictions from the hygrothermal bed models BED and Lectus with each other, and against fieldwork data. The results showed that both models predict measurement results satisfactorily, once the uncertainties due to input variables and error measurement are taken into account. In particular, the BED model predicts the surface temperatures slightly better than the Lectus model (R-squared value – BED: 0.74, Lectus: 0.72). However, Lectus RH surface predictions match more closely the measured results, than the BED predictions (R-squared value – BED: 0.63, Lectus: 0.74). The results also show that it is possible to calculate conditions within the mattress depth from the BED predictions, which match the measurements (and the Lectus predictions) satisfactorily – once uncertainties are taken into account.

The predictions from the BED and the Lectus models for (average) conditions within the mattress (under the chest area) are not dissimilar, and they both match measurement results - once the uncertainties are taken into account. Therefore, it could be concluded that the BED model (which is simpler) could be utilised for scenarios modelling of HDM growth conditions. However, Popmite with transient input conditions predicts the fieldwork results (mite bags) better than when the MPI model or when constant conditions are utilised. Consequently, it may still be preferable to utilise Lectus for scenarios modelling – unless conditions with very low RHs are being considered.

Chapter 10: References

Biddulph P, Crowther D, Leung B, Wilkinson T, Hart B, Oreszczyn T, Pretlove S, Ridley I, Ucci M, (2007), Predicting the population dynamics of the house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle, *Experimental and Applied Acarology*, 41(1-2): 61-86.

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

BSI, (2002), *BS EN ISO 13788:2002. Hygrothermal performance of building components and building elements. Internal surface temperature to avoid critical surface humidity and interstitial condensation. Calculation methods.* London: BSI.

Hart BJ, Crowther D, Wilkinson T, Biddulph P, Ucci M, Pretlove S, Ridley I and Oreszczyn T, (2007), Reproduction and development of laboratory and wild house dust mites (*Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae)) and their relation to the natural dust ecosystem, *Journal of Medical Entomology*, 44(4): 568-574.

Pretlove SEC, Oreszczyn T, Ridley I, Wilkinson T, Crowther D, (2005), A steady-state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements, *Building Services Engineering Research and Technology*, 26(4): 301-314.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (submitted), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air.*

CHAPTER 11:

USE OF THE MODELS IN A PILOT INTERVENTION STUDY

CHAPTER 11: USE OF THE MODELS IN A PILOT INTERVENTION STUDY

11.1 Introduction

The most obvious application for the models tested in this thesis are desk-based scenarios modelling studies, aimed at identifying those building features and occupant behaviours which are most crucial for mite growth/decline in beds. This scenarios are modelled in Chapter 12. However, potentially the models can also be utilised in fieldwork studies, for example in intervention studies aimed at reducing mite levels (and asthma symptoms). Chapter 1 and 2 identified the complexity of undertaking such studies, and models such as those tested in this thesis can help interpret the findings. This chapter discusses a pilot intervention study on the effectiveness of house dust mite allergen avoidance for twelve asthmatic children (two being controls). In addition to mite allergen removal, the study included tailored advice aimed at reducing mite population growth via changes in moisture production, heating and ventilation habits. Consequently, the study addressed four issues: 1) the effect of allergen removal on the children's health; 2) the effect of tailored advice on occupant behaviour and the resultant hygrothermal conditions; 3) the effect of the hygrothermal changes on mite populations; and 4) the efficacy of monitoring/modelling techniques. This chapter mainly focuses on the last 3 issues, although some aspects of the health impacts are also briefly discussed.

Due to the short time-scale and small sample size associated with this study, this chapter does not aim to prove the clinical efficacy of allergen avoidance, nor the effectiveness of psychrometric (behavioural) control methods for dust mites. Instead, this chapter discusses the advantages and disadvantages of the methodology adopted in the pilot study, which could be used on a larger scale study. The role of the models tested in this thesis is also discussed.

The next section (11.2) describes the study design and the methodology utilised in the pilot study. Section 11.3 illustrates the baseline results, and section 11.4 describes the tailored advice provided to each household, for changing hygrothermal behaviours. Section 11.5 describes the post-intervention results, and

section 11.6 discusses the role of modelling techniques in the study. The chapter ends with a summary discussion (section 11.7).

11.2 Study design and methodology

This section illustrates the study design and the methodology utilised in the pilot study. The study aimed to test a methodology for the reduction of asthma and allergic rhinitis symptoms in children sensitised to house dust mites (HDM), through allergen avoidance measures.

Psychrometric HDM control measures (i.e. reducing mite numbers through the control of temperature and RH) do not remove any existing allergen reservoirs, which can be rather long-lasting (see Chapter 2). Therefore, in any study aiming at reducing respiratory health symptoms, any pre-existing HDM allergens have to be removed. However, most allergen removal strategies (e.g. steam-cleaning) are time-consuming, they cannot be applied to all possible dust reservoirs, and they need to be repeated over time. This is because if hygrothermal conditions are favourable to mite growth, Der p1 levels will be replenished after some time. Therefore, psychrometric control methods should complement allergen removal measures, since they potentially strengthen and extend their effects.

Most studies on the psychrometric control of house dust mites in housing have focused on mechanical ventilation (e.g. Howieson, 2003). However, there is some scope for modifying residential hygrothermal conditions by changing the occupants' heating and ventilation habits. For example, a well-designed extractor fan can remove up to 70% of moisture generated during cooking (Liddament, 2001). Also, a UK study found that the presence of an extractor fan in the kitchen was associated with lower HDM allergen concentrations (Luczynska *et al*, 1998). Furthermore, a large scale study concluded that mite allergen exposure may be reduced by increasing the ventilation of the bedroom, particularly in winter (Zock *et al.*, 2006). Nevertheless, very few intervention studies have been carried out attempting to reduce HDM levels through the modification of occupant behaviour alone. Compared with methods acting on the building fabric or on heating and ventilation systems, behavioural changes can be inexpensive and implemented in

shorter time-scales. These were the main reasons for testing this psychrometric control method in the pilot study described in this chapter.

The study illustrated in this chapter was filmed by *Twenty Twenty Television* and resulted in two 50-minutes episodes of the UK TV programme series 'Dispatches' on Channel 4 in April 2006 (Coates, 2006), with an aim to illustrate to TV viewers the potential benefits of allergen avoidance. As previously mentioned, due to its short time-scale and small sample size, the intervention study did not aim to establish the clinical efficacy of allergen avoidance, but rather give the researchers the opportunity to test the study protocol for a larger future study.

In October 2005, twelve asthmatic mite-sensitive children aged 6 to14 were selected in the London area by the TV production team. Eleven dwellings/households were examined overall, since two of the children were siblings living in 1 dwelling (here referred to as bedroom/child 12a and 12b). Before the interventions were implemented, a pre-intervention study was carried out in November 2005, where baseline measurements were taken of: the children's health status, HDM numbers and allergen levels in each dwelling, as well as hygrothermal conditions, building characteristics, and heating and ventilation habits. The children's asthma and health status was assessed by Dr Glenis Scadding, consultant physician at the Royal National Throat Nose & Ear Hospital. The initial medical examination included: interview on the medical history; ear, nose and throat examination; skin prick test (for HDM, moulds, cat, dog, grass pollen); spirometry test (to assess lung function); and upper and lower airway nitric oxide. A number of interventions were then carried out, followed by a post-intervention study, where the children's health and the dwellings' hygrothermal conditions were monitored for 6 weeks (Dec 05-Jan 06).

Dust samples for the determination of mite allergen levels (Der p1) and of live mite numbers were taken at the beginning of the pre-intervention period (baseline) in the child's bedroom (mattress, pillow, one soft toy and floor) and the living room (floor and sofa). Samples were also taken at the *start* of the post-intervention period (after intensive cleaning), and at the *end* of the post-intervention period (i.e. approximately 6 weeks after the interventions). For each dust sample, 1 m^2 was vacuumed for 2 minutes, using a Mitest dust collector (Indoor Biotechnologies, Cardiff, UK) attached to a Bosch Arriva vacuum cleaner

(1400 W). The mattresses and pillows were stripped of bed linen before the dust collection. The counting of live mites was performed by a trained acarologist. In addition, extracts of sieved dust samples were assayed for Der p1, using ELISA (Indoor Biotechnologies, Cardiff, UK). The lower limit of detection was 3 ng/ml. Samples below this limit were assigned the detection limit value. During the pre-intervention study, temperature and relative humidity were monitored for approximately 2 weeks in November 2005, with TinyTag dataloggers (*geminidataloggers.com*) logging at 15 minutes intervals at 4 locations in each dwelling (child's bedroom, living room, kitchen, bathroom and outdoors). A detailed survey of the dwelling was also carried out, which included a fan-pressurisation test (Retrotec, Q5AE, High Power) and a thermal imaging analysis of the building envelope via an infrared camera (Flir, S65 HS)[1]. These were aimed at identifying any specific problems in the building fabric and to tailor the advice on heating and ventilation habits. A questionnaire was administered to the child's parents, to assess their existing heating, ventilation and moisture-producing habits.

After the baseline measurements, the following interventions were carried out: professional steam-cleaning of the child's bedroom and thorough cleaning of the dwelling; replacement of carpets in the child's bedroom with laminate flooring; covering mattresses, pillows and duvets with micro-porous mite-proof barriers; removing pets and cuddly toys; and avoiding exposure to environmental tobacco smoke. The participants were also advised to implement a thorough cleaning regime throughout the post-intervention period.

Following the analysis of the pre-intervention study results, tailored advice was also provided on moisture production, heating and ventilation. The main aim of the advice was to ensure the reduction of indoor relative humidity levels. Since most dust mite allergen and live mites had been removed during the steam-cleaning, any changes in hygrothermal conditions due to the implementation of the tailored advice were not intended to have an *immediate* impact on mite levels. Rather, the psychrometric changes aimed to support and sustain over time the effect of allergen removal. Temperature and relative humidity were monitored

[1] The fan-pressurisation test and the thermal imaging were carried out by Dr Dejan Mumovic (UCL) and by Dr Stephen Pretlove (Kingston University), who also contributed to the initial data analysis and to formulation of the tailored advice given to the participants.

during the post-intervention period, in order to assess the efficacy of the advice. Outdoor hygrothermal conditions were also monitored throughout the study.

Since most existing mites had been removed during the steam-cleaning, it was not possible to study the *direct* impact of hygrothermal conditions on mite populations. Therefore, the mite bags technique (see Chapter 4) was adopted during the post-intervention study, whereby 20 live 'wild' DP mites were caged with food in a mite and allergen proof 'bag' made from mite-proof material. The 'mite bags' were placed in each dwelling at the *beginning* of the post-intervention period, in the child's bedroom (next to the hygrothermal datalogger) and in the child's bed (under the bedding, corresponding to the feet area) - where a sensor was also positioned. The loggers utilised in the beds were the same as those utilised in the Series 1 study (see Chapter 4). Three replicate mite bags were placed in each location. In those dwellings which appeared more at risk from mite growth – based on the modelling results, see section 11.6 – additional mite bags and sensors were positioned in the child's bed, under the pillow and under the chest area (Dwelling: 1,7, 10, 11, 12). The mite bags were placed on the top surface of the mattress, which was then covered with a mite-proof cover[2]. An additional padded cover[3] was also placed on top of the mite-proof cover, in order to reduce any discomfort experienced by the children due to the sensors, and in order to prevent the sensor from breaking. In addition, it was anticipated that the extra padded cover might protect the mite bags from the body's heat, and therefore give the mites a greater survival chance (most mites in the mattress surface under the chest had died in Series 1). After six weeks the bags were retrieved and the live mites counted. This gave an indication of the likely impact of the dwelling's hygrothermal conditions on mite populations. Ideally, two different sets of mite bags should have been placed in each household, once during the pre-intervention study, and once during the post-intervention study. However, for practical reasons[4] the pre-intervention study could only be carried out for 2 weeks, which was considered an insufficient time for the mite bags to be in place. Consequently, the mite bags were only utilised during the post-

[2] Diagenics micro-porous mite proof bed cover, water vapour permeable (www.diagenics.co.uk).
[3] John Lewis: Jonelle mattress quilted cover, with 100% cotton cover and 100% polyester filling, code: 607/40202.
[4] Time constraints for the TV production team.

intervention study. Although this did not allow for a *direct* comparison of mite growth in the bags before and after the interventions, modelling technique were adopted to overcome this problem (see section 11.6). Furthermore, the mite bags gave an indication of those dwellings still at risk from mite growth, after the interventions. It should be mentioned that the results from the mite bags utilised in the pilot were also adopted to test the validity of the population models (Chapter 7 and 8).

For the two households acting as controls (dwelling 6 and 8), the interventions were carried out *at the end* of the post-intervention period, but their dwelling's hygrothermal conditions were monitored throughout the study. The health of all children was monitored during the study - through a combination of questionnaires and self-administered peak flow tests – and a final medical examination was carried out at the end of the post-intervention study.

Throughout the study, the author of this thesis played an active role in the research team– from the study design phase, to the implementation and data analysis. The author carried out the dwelling and participant surveys – in collaboration with Dr Mumovic (UCL) and Dr Pretlove (Kingston University). The author also gathered the dust sample and installed most loggers (or liaised with the TV researchers for loggers installation), as well as the mite bags, which were produced by Toby Wilkinson (Cambridge University). The caged mites at the end of the monitoring period were counted by Dr Barbara Hart (Royal Agricultural College), whilst the allergen assays were carried out by the company Acaris (www.acaris.co.uk). The tailored advice was formulated by the author, in conjunction with Prof Oreszczyn (UCL) and Dr Pretlove. Dr Biddulph (UCL) did some preliminary analysis of the mite bag results. The data analysis presented in this chapter was carried out by the author. The next section describes the baseline results, from the pre-intervention study. The study was published on the BSERT Journal as a Technical Note (Ucci *et al.*, 2007, see Appendix A.0.4).

11.3 Pre-intervention study: baseline results

This section illustrates the baseline results, from the pre-intervention study. In particular, the following pre-intervention study results are presented: mite infestation levels, building characteristics and hygrothermal conditions.

Table 11.3.1 shows the Der p1 allergen results. Approximately a quarter (26%) of all samples were below the allergen detection limit. Pillows had the highest geometric[5] mean of Der p1 concentration, followed by living room floor and toys. However, allergen concentrations can be misleading, since little dust was found in toys and pillows. Therefore, the results were also expressed in terms of 'allergen load': total allergen weight collected for a given vacuumed area, corresponding to μg Der p1/m^2 (μg Der p1/total object area, for pillows and toys).

Table 11.3.1 Baseline Der p1 allergen results

	Allergen Concentration (Der p1 μg/g)					Allergen Load (Der p1 μg/m²)*				
Bedroom	**M**	**P**	**BF**	**LF**	**T**	**M**	**P**	**BF**	**LF**	**T**
1	1.49	63.39	4.22	2.42	18.21	1.30	0.44	1.73	0.80	0.04
2	0.13	4.48	0.50	(–)	9.47	0.16	0.04	0.11	(–)	0.09
3	0.09	0.62	0.15	(–)	1.67	0.10	0.13	0.15	(–)	0.02
5	0.05#	1.16	0.05	(–)	0.05#	0.01#	0.01#	0.01#	(–)	0.01#
6c	(–)	0.34	0.21	(–)	0.05#	0.02	0.08	0.24	(–)	0.01#
7	4.19	56.87	3.14	1.80	(–)	2.79	2.57	1.54	1.12	(–)
8c	2.05	7.39	0.57	5.47	7.34	0.29	0.21	0.23	0.34	0.18
9	(–)	(–)	(–)	(–)	(–)	0.01#	0.01	0.05	(–)	0.01#
10	0.05#	2.82	0.05#	2.90	68.56	0.01#	0.31	0.01#	1.62	0.23
11	0.46	4.47	0.34	(–)	(–)	0.24	0.24	0.44	(–)	(–)
12a	0.11	6.33	0.16	1.44	(–)	0.02	0.62	0.13	1.58	(–)
12b	(–)	0.09	0.19	(1.44)	(–)	(–)	0.07	0.17	(1.58)	(–)
Geom. Mean	**0.30**	**3.05**	**0.33**	**2.51**	**2.32**	**0.07**	**0.12**	**0.13**	**0.95**	**0.03**

M=Mattress; P=Pillow; BF=Bedroom Floor; LF=Living Room Floor; T=Soft Toy; cControl Dwelling; *In pillows the allergen load refers to the allergen weight over the *total* pillow area; #Below allergen detection limit; (–) Missing data.

It was found that in this study 8 out of 11 dwellings (73%) had a least one dust reservoir with Der p1 concentrations greater than 2 μg Der p1/g (often referred to as the "WHO threshold for HDM sensitisation": Platts Mills and de Weck, 1989) and 3 out of 11 dwellings (27%) had a least one dust reservoir with concentrations

[5] The geometric means was utilised since the data was skewed. In fact, most published information on Der p1 levels is reported in terms of geometric means (e.g. see Table 2.3.2 of Chapter 2, Literature Review).

greater than 10 μg Der p1/g (often referred to as the "WHO threshold for asthma exacerbation": Platts Mills and de Weck, 1989). In 82% of all dust samples no live mites were detected, which was not surprising because of the seasonal nature of mite populations (Crowther *et al.*, 2006). Furthermore, it is notoriously difficult to vacuum live mites, which can cling to fibres and avoid being removed. Live mite numbers were higher on average in living room floors, followed by bedroom floors. Dwellings 1 and 7 had by far the highest average allergen concentrations and allergen loads, and high live mite levels (Table 11.3.1 and 11.3.2).

The twelve properties that were visited for the study included: 5 flats, 1 detached house and 6 terraced houses, ranging from a large three storey Edwardian terrace to a 1980s purpose-made flat. Table 11.3.2 illustrates the baseline hygrothermal measurements. The daily moisture production (kg/day) was estimated by using the questionnaire results and the moisture algorithm of Condensation Targeter II (Oreszczyn and Pretlove, 1999). Dwelling 7 had the highest estimated moisture production (13.7 kg/day), due to high occupancy levels. The fan-pressurisation test results at 50 Pa were converted to an estimated air-infiltration rate in air changes per hour under average external conditions (Sherman, 1998). Dwelling 1 had the lowest air-infiltration rate (0.24 ach^{-1}) – mostly because of its small exposed surface area. Dwelling 1 also had the highest average RH, as well as the highest average bedroom vapour pressure excess (VPX: Bedroom Vapour Pressure minus Outdoor Vapour Pressure).

The Critical Equilibrium Humidity (CEH) for each monitored time step was also calculated as a function of temperature, using data for the mite Dermatophagoides Farinae (DF) (Arlian and Veselica, 1981). In the UK this species is not as common as the Dermatophagoides Pteronyssinus (DP). Although there is some evidence to suggest that the CEH for DP mites may also be temperature dependant (Crowther *et al.*, 2006), there is insufficient data to establish this relationship fully, and therefore the equation developed by Arlian and Veselica (1981) for DF mites had to be utilised in this study. The results showed that on average the bedrooms RH exceeded CEH for 57% of the monitored period, and in the bedrooms of Dwelling 1, 11 and 12 this figure was 90% or more.

Table 11.3.2 Baseline measurements for building characteristics, hygrothermal conditions and mite infestation levels.

Dwelling	[θ]Moisture Product. (kg/day)	Volume (m^3)	[Δ]Air Infiltr. (ach^{-1})	*Mites (mites/ m^2)	*Der p1 Conc. (µg/g)	#Pre, VPX (kPa)	#Pre, % Time CEH>RH (%)	#Pre, Temp (°C)	#Pre, RH (%)
1	7.2	163.1	0.2	20.3	23.0	0.6	92.1	20.9	68.7
2	4.2	198.6	0.4	0.0	1.7	0.2	18.1	20.7	52.3
3	3.4	127.2	0.5	0.0	0.3	0.4	66.8	20.8	57.3
5	6.5	484.5	0.9	0.0	0.4	0.2	0.0	20.9	40.4
6[c]	11.9	286.2	1.1	21.7	0.3	0.3	36.3	20.6	54.1
7	13.7	189.8	0.6	17.7	21.4	0.2	73.4	18.7	57.5
8[c]	6.4	137.4	0.5	0.0	3.3	0.4	37.4	22.3	55.8
9	7.9	215.9	1.4	0.0	(-)	0.2	47.2	20.2	54.5
10	6.3	141.3	0.6	0.0	0.9	0.4	20.2	22.1	54.6
11	10.1	396.1	1.3	5.3	1.8	0.2	99.2	17.4	61.8
12a	5.3	263.0	0.6	1.3	2.2	0.3	90.3	(17.6)	57.4
12b	5.3	263.0	0.6	0.0	0.8	0.3	100.0	(17.4)	60.5
Average	**7.7**	**227.3**	**0.7**	**5.1**	**1.4[α]**	**0.3**	**56.8**	**20.5**	**55.8**
Outdoor Conditions								**11.4**	**76.8**

[c]Control Dwelling; *Bedroom, Average of: Mattress, Floor, Pillow; #Child Bedroom; [θ]Whole dwelling, estimated; [α]Geometric Mean; [Δ](Air-infiltration measured at 50 Pa)/20; (-) Missing data. Note: central heating in Dwelling 12 was malfunctioning in the pre-intervention study.

This section presented the baseline pre-intervention results. The following section illustrates the tailored advice on hygrothermal behaviours.

11.4 The tailored advice for behavioural hygrothermal changes

This section describes the tailored advice which was provided to each household on the most appropriate heating, ventilation and moisture-production patterns, which could reduce house dust mite populations. The tailored advice was based on the findings of the pre-intervention study. For example, in a leaky dwelling inhabited by a household with average moisture production, it is not advisable to increase ventilation rates further, since this might excessively reduce temperature levels. Although low indoor temperatures increase mite egg-to-adult development times, they also result in higher RH values. Therefore, depending on the dwelling and occupant behaviour characteristics, each household was advised to implement one, or a combination of, the following measures: a) reducing moisture production; b) increasing ventilation levels; and c) increasing temperature levels.

For example, Household 1 – which had the smaller air infiltration rates, and highest RH and VPX levels – was advised to:

- Only dry clothes indoors in a well ventilated room, which is closed to the rest of the home;
- Use the extract fans in the kitchen and the bathroom during occupation, and for at least 15 minutes afterwards;
- Keep the trickle vents always open;
- Leave the windows always slightly open. If this is not possible (e.g. for security reasons), the windows should be left open for 30 minutes in the morning, and 30 minutes in late afternoon. When doing this, the windows do not need to be fully opened, particularly if it is a cold day.

On the other hand, Household 11 - which had low temperatures and high infiltration rates - was advised to increase indoor temperatures either by increasing the set-point temperature on the thermostat or by increasing the period that the heating is on.

Households 2 and 9 were advised not to change their behaviour significantly in order to maintain their current RH levels, which appeared satisfactory in the pre-intervention study. The control households (6 and 8) did not receive the advice until the end of the whole study.

The following section describes the post-intervention results.

11.5 Post-intervention results

This section illustrates the post-intervention results. In particular, the hygrothermal changes and the mite bags results are described. The impact of allergen reduction on the children's respiratory symptoms is also briefly discussed.

Table 11.5.1 shows the post-intervention results for the hygrothermal conditions, including the difference with pre-intervention results.

Table 11.5.1 Post-intervention hygrothermal results, and difference with pre-intervention conditions, for the child's bedroom.

Bedroom Number	Post: VPX (kPa)	Difference: Pre-Post VPX (kPa)	Post: Temp. (°C)	Difference: Pre-Post Temp. (°C)	Post: RH (%)	Difference: Pre-Post RH (%)	Post: % Time CEH>RH	Difference: Pre-Post % Time CEH>RH (%)
1	0.6	0.0	19.4	1.5	59.8	8.9	76.3	15.8
2	0.1	0.1	19.5	1.2	40.7	11.6	0.0	18.1
3	0.2	0.2	19.6	1.2	41.9	15.4	0.0	66.8
5	0.0	0.2	19.2	1.7	37.6	2.8	0.0	0.0
6	0.1	0.2	18.0	2.6	41.6	12.5	1.8	34.5
7	0.2	0.0	17.4	1.3	47.4	10.1	9.5	63.8
8	0.5	-0.1	21.9	0.4	48.4	7.4	0.1	37.3
9	0.2	0	21.3	-1.1	38.4	16.1	0.0	47.2
10	0.3	0.1	20.4	1.7	43.5	11.1	1.2	19.0
11	0.2	0	17.8	-0.4	47.8	14.0	12.1	87.1
12a	0.0	0.3	17.3	0.3	40.3	17.1	5.6	84.7
12b	0.2	0.1	17.2	0.2	47.1	13.4	2.8	97.2
Average*	**0.2**	**0.1**	**18.9**	**0.8**	**44.5**	**12.1**	**10.8**	**50.0**
Outdoor	**(n.a.)**	**(n.a.)**	**6.6**	**4.8**	**80.2**	**-3.4**	**(n.a.)**	**(n.a.)**

*Excluding controls (bedroom 6 and 8)

The results show that in most dwellings there was a small reduction in the average indoor temperatures from the pre to the post intervention periods. This was due to the average outdoor temperature dropping from 11.4 $^{\circ}$C (pre-intervention) to 6.6 ^{0}C (post-intervention). Dwelling 11 (which was specifically advised to increase the temperature levels) and Dwelling 7 (advised not to change their habits) experienced a small increase in indoor temperatures. The VPX in the bedrooms decreased on average by 0.1 kPa in all the properties (average excluding the controls). However, the VPX *increased* in the control Dwelling 8 and remained largely unchanged in Dwellings: 1, 7, 9, 11. In all dwellings, the bedroom RH decreased from the pre to the post intervention periods. Furthermore, in all dwellings the percentage of time the bedroom RH was greater than CEH ('% time RH>CEH') also decreased, with the largest difference from pre to post values in bedrooms: 12a, 12b and 11. In Bedroom 3, the '% time RH> CEH' was reduced from 66.8% in the pre-intervention, to 0% to the post-intervention. On the other hand, despite a reduction of 15.8% (Pre-Post difference), in Bedroom 1 the '% time RH> CEH' was still 76.3% in the post-intervention study.

It should be highlighted that the reduction in indoor RH levels was partly due to outdoor conditions. Although the average outdoor RH increased from 76.8% to 80.2%, the post-intervention outdoor absolute humidity was lower. Therefore, in

order to disentangle the weather effect from the advice implementation, the pre and post intervention RHs were adjusted for each bedroom. It was assumed that if the pre and post intervention period had exactly the same weather conditions, the indoor temperatures would be the same, and the indoor RH would be dependent on the dwelling's vapour pressure excess, as well as on the outdoor vapour pressure. Since the outdoor conditions had been monitored for a longer period during the post intervention period, the indoor RHs were adjusted in relation to the post intervention weather conditions. The adjusted vapour pressure was calculated as follows:

$$Pre\_VP^{Adj} = Out\_VP + Pre\_VPX \quad [11.1]$$

$$Post\_VP^{Adj.} = Out\_VP + Post\_VPX \quad [11.2]$$

where Out_VP is the outdoor vapour pressure monitored during the post intervention study; Pre_VPX is the average vapour pressure excess measured during the pre intervention period; Post_VPX is the average vapour pressure excess measured during the post intervention period. Figure 11.5.1 illustrates a schematic representation of the adjustment calculation for the vapour pressure.

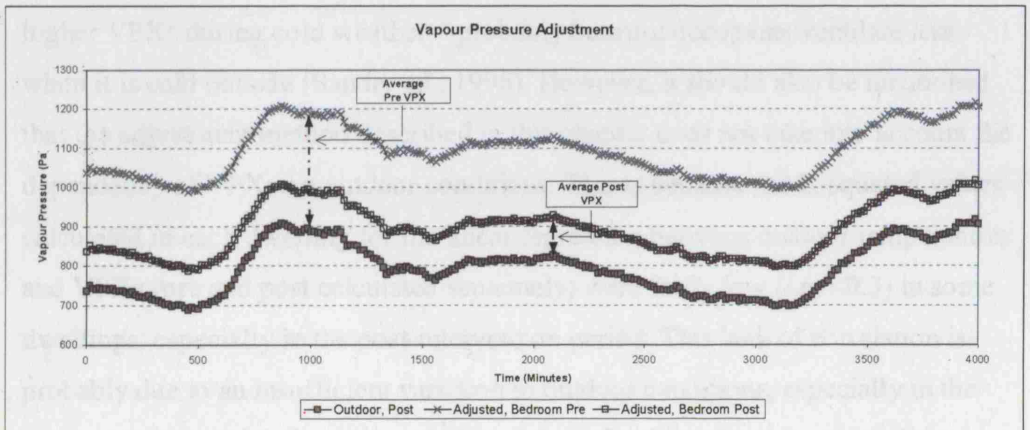


Fig 11.5.1 Schematic representation of the adjustment calculation for the vapour pressures, pre and post intervention.

Once the adjusted VPX was calculated, the adjusted RH was then calculated as follows:

$$Pre\_RH^{Adj} = RH(Post\_T, Pre\_VP^{Adj}) \quad [11.3]$$

$$Post\_RH^{Adj} = RH(Post\_T, Post\_VP^{Adj}) \quad [11.4]$$

where Pre_RH^{Adj} is the adjusted pre intervention RH, as a function of the monitored post temperature (Post_T), and the adjusted pre vapour pressure (Pre_VP^{Adj}). The $Post\_RH^{Adj}$ is the adjusted post intervention RH, as a function of the monitored post temperature (Post_T), and the adjusted post vapour pressure ($Post\_VP^{Adj}$).

The adjustment method described so far was developed in relation to the data available to this study. Previous studies have adopted other methods, which were however considered unsuitable for this study. For example, in the Warmfront study a standardised indoor temperature was calculated for each dwelling, corresponding to an outside temperature of 5 °C, by regressing the indoor temperatures on the outdoor temperatures (Oreszczyn *et al.*, 2006). However, in this pilot study it was considered necessary for modelling purposes[6] to obtain a *dataset* of adjusted conditions, as opposed to *one* normalised value for each dwelling. Furthermore, the dissimilarities in outdoor conditions between the pre and the post intervention periods in this study might have confounded the Warmfront method, since outdoor conditions can affect hygrothermal behaviours. For example, the VPX in dwellings can be dependent on outdoor conditions, with higher VPXs during cold weather – probably because occupants ventilate less when it is cold outside (Sander ed., 1996). However, it should also be mentioned that the adjustment method described in this chapter does not take into account the dependency of VPXs on outdoor conditions. This is because the R-squared values calculated in each dwelling for the linear regression between outdoor temperatures and VPXs (pre and post calculated separately) were fairly low (i.e. <0.3) in some dwellings, especially in the post-intervention period. This lack of correlation is probably due to an insufficient variation in outdoor conditions, especially in the pre-intervention period, which only lasted 2 weeks. Therefore, future studies should ideally monitor the pre-intervention conditions for a whole season (or preferably a year), and then monitor the post-intervention conditions for the same length of time.

[6] Modelling conditions in the mite bags, see Section 11.6

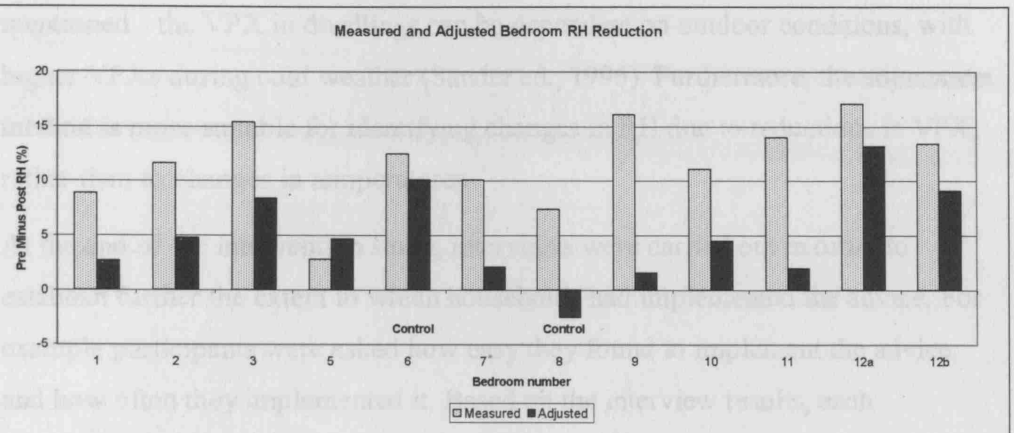


Fig 11.5.2 Comparison between measured and adjusted reduction in bedroom RHs.

Figure 11.5.2 shows the reduction (difference) in the measured and in the adjusted bedroom RHs. The results show that once the impact of changes in outdoor conditions is taken into account through the adjustment procedure described earlier, the reduction in bedroom RHs is smaller than measured results, particularly for some bedrooms. The average adjusted reduction in the bedrooms RH was 5.1% (pre-post difference in average RH). A paired T-test was carried out, in order to assess whether there was a statistically significant difference between pre and post bedroom RHs. The results were statistically significant ($p<0.01$) for both the *measured* pre and post RHs, and the *adjusted* pre and post RHs.

The importance of the adjustment procedure is highlighted by the case of the control Dwelling 8, where the measured bedroom RH decreased from the pre to the post intervention period. However, its adjusted RH *increased* (by a small amount). This was due to an increase in the actual post-intervention vapour pressure excess (Table 11.5.1), probably because of a reduction in window opening for the colder outdoor temperatures. It should also be highlighted that the other control dwelling (Dwelling 6) experienced an above average reduction in adjusted RH levels. However, it is possible that Household 6 modified its behaviour, as a result of participating to the study.

It should be mentioned that the adjustment calculations described so far are likely to underestimate the RH reductions due to the advice. This is because - as already

mentioned - the VPX in dwellings can be dependent on outdoor conditions, with higher VPXs during cold weather (Sander ed., 1996). Furthermore, the adjustment method is more suitable for identifying changes in RH due to reductions in VPX, rather than to changes in temperatures.

At the end of the intervention study, interviews were carried out in order to establish further the extent to which households had implemented the advice. For example participants were asked how easy they found to implement the advice, and how often they implemented it. Based on the interview results, each household was given an 'implementation score' by the interviewer. No correlation was found between the measured pre/post RH reduction and the 'implementation score'. Therefore, for those dwellings which experienced small reductions in RH, it is difficult to establish whether this was due to: a) lack of participants' action, b) adverse building characteristics hindering changes, c) limitations of the advice itself. However, it also became apparent that occupants experienced some difficulties in reporting their ventilation habits coherently. Measuring ventilation rates and a bigger sample size would make it easier to assess the effect of the advice on occupant behaviour, as well as on hygrothermal conditions.

Table 11.5.2 illustrates the mite cages results and it shows that in most dwellings the mite population declined. This is not surprising, considering the time of year. The location under the pillow was the most favourable for mite growth, probably due to the moisture exhaled whilst breathing during sleep. In Dwelling 1 - which in the post-intervention study had the highest average RH and the highest percentage of time the bedroom RH > CEH (Table 11.5.1) - the final population in the mite bags was greater than the initial one (20 mites). This also proves that the mites can grow even in caged conditions, if the hygrothermal conditions are favourable.

Table 11.5.2 Remaining live mites in sealed 'mite cages' after 6 weeks (end of post intervention period)

Bedroom Number	Live mites after 6 weeks (average of 3 mite bags)*			
	Bedroom	Bed: Under Pillow	Bed: Under Chest	Bed: Under Feet
1	26.3	35.0	18.7	7.3
2	(missing)	(–)	(–)	2.0
3	0.0	(–)	(–)	0.0
5	1.3	(–)	(–)	0.0
6	2.7	(–)	(–)	(–)
7	4.0	11.7	0.7	0.0
8	0.7	(–)	(–)	(–)
9	0.0	(–)	(–)	0.0
10	0.3	9.0	0.0	2.0
11	7.3	24.7	10.7	0.3
12a	2.7	1.7	1.7	0.3
12b	0.0	8.0	2.0	5.3

* Starting population in each mite bag: 20 adult DP mites; (–) Mite cages not installed.

As already mentioned, at the beginning of the intervention period all soft furnishings were steam-cleaned. As a result, a statistically significant reduction was found for Der p1 load levels ($p<0.01$). At the end of the post-intervention period, the initial medical examination was repeated, and a 'symptoms improvement score' was determined for each child[7]. Figure 11.5.3 shows the child's improvement score plotted against the reduction in the bedroom allergen load (difference between pre and post intervention measured allergen loads). The graph suggests that a (weak) correlation exists between health improvement and allergen reduction ($r=0.55$; $R^2=0.30$). Some children experienced an improvement which did not correspond to a dramatic reduction in Der p1 levels. This may be due to confounding variables, such as concomitant allergies or placebo effect.

[7] The health improvement score was determined by Dr Glenis Scadding, based on medical examinations.

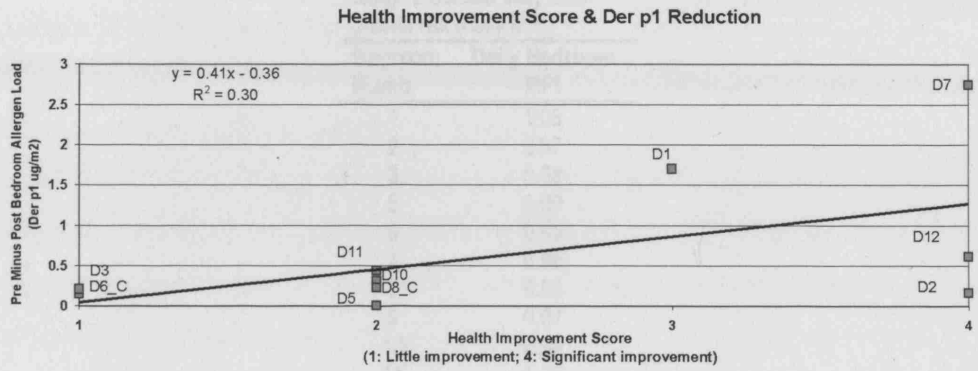


Fig 11.5.3 Respiratory symptoms 'improvement score' and reduction of Der p1 levels in child's bedroom.

The following section illustrates the role of the hygrothermal population models in the pilot study.

11.6 The role of modelling techniques

This section describes the use of the population models in the pilot study. The models were adopted in the study in order to:

- Help identifying those dwellings most at risk from mite infestation in the pre-intervention study;
- Assess the effect of changes in hygrothermal conditions on mite populations.

In order to identify those dwellings most at risk of mite infestation, the average hygrothermal conditions measured in the children's bedrooms during the pre-intervention study were utilised in the MPI model, whose output is the MPI index - where for example 1.1 indicates 10% population growth and 0.9 indicates 10% population decline. The MPI output corresponds to the Mite Population Index after *21 days*, for given steady-state hygrothermal conditions. Since the pre-intervention study was approximately 2 weeks (with small changes between dwellings) and in order to allow the comparison between the various bedrooms, the predicted *daily* MPI value was calculated (see Chapter 8, equation 8.1). The results are illustrated in Table 11.6.1.

Table 11.6.1 Pre-intervention study: predicted daily mite-growth risk (daily MPI)

Bedroom Numb.	Daily Bedroom MPI
1	1.03
2	0.97
3	0.98
5	1.00
6	0.95
7	0.98
8	0.95
9	0.97
10	0.98
11	1.00
12a	1.00
12b	0.99
Average	**0.98**

The results indicate that during the pre-intervention period the mite populations were rather stable (average MPI $\cong$ 1), suggesting that even small hygrothermal changes could determine whether the population grows or declines. Bedroom 1 had the highest predicted daily population growth. This is in agreement with the results from the dust samples. The other bedrooms with a higher than average MPI index were: 5, 12a, 12b and 11. These bedrooms did not have the highest mite levels within the sample (Table 11.3.2). This is probably due to a reservoir effect: for example, allergen levels are higher in older mattresses and might also depend on cleaning regimes, floor types, etc. (see Chapter 2).

In this study the identification of those dwellings most at risk from mite growth determined which dwellings should have extra sets of mite bags, as a limited number of mite bags and equipment was available (see previous section). In this study the mite bags were utilised mostly to assess the impact of post-intervention hygrothermal conditions on mite growth. However, additional aims for the mite bags were: a) to help test the population models (Chapter 7), and b) to assess where in the bed mites might flourish. Since the mite bags are time-consuming to produce, it was decided to install the extra sets of mite bags only in those dwellings most at risk from mite growth, based on the MPI predictions. However, the identification of those dwellings most at risk of mite growth can be very useful in other types of intervention studies or in remedial projects, for example in order to prioritise the remedial works.

The other role of the population models in this study was to assess the effect of changes in hygrothermal conditions on mite populations. In particular, it was already highlighted that due to the allergen and mite removal, it was not possible to test the direct impact of changes in hygrothermal conditions on mite populations. The mite bags were utilised for this purpose. However, due to practical reasons, the pre intervention study could only last for two weeks, as opposed to the 6 weeks of the post intervention study. Therefore, a direct comparison in mite bags results between the pre and the post intervention studies would not have been possible, and consequently no mite bags were utilised in the pre intervention study. In any case, a *direct* comparison would have been based on the effect of *actual* hygrothermal conditions, which included the confounding variable of changes in outdoor conditions. Therefore, in this study Popmite was utilised in order to model the effect of *adjusted* pre and post hygrothermal conditions on the mite bags populations. The adjusted pre and post conditions were calculated as described in previous section. These conditions were then adopted in Popmite, with a starting population of 20 adult mites. The results are summarised in Table 11.6.2.

Table 11.6.2 Popmite predictions for the *adjusted* pre and post hygrothermal conditions

Bedroom Number	Pre Minus Post Adjusted Average Bedroom RH (%)	Pre: Total Number of Predicted Mites (incl. eggs)	Post: Total Number of Predicted Mites (incl. eggs)	Ratio: Post/Pre Total Predicted Mites (incl. eggs)
1	2.7	59.8	55.0	0.9
2	4.8	13.3	0.0	0.0
3	8.4	36.5	0.0	0.0
5	4.7	10.6	9.0	0.8
6 (control)	10.1	23.9	12.4	0.5
7	2.1	25.7	20.7	0.8
8 (control)	-2.5	7.8	12.8	1.6
9	1.6	0.4	0.0	0.0
10	4.5	14.0	11.5	0.8
11	2	18.2	14.9	0.8
12a	13.2	20.5	10.0	0.5
12b	9.1	30.5	14.9	0.5

The results show that in all bedrooms except the control Bedroom 8, there is a reduction in the predicted number of mites in the post intervention, based on the adjusted hygrothermal conditions. This suggests that all dwellings implemented the advice to a sufficient level for mite growth reduction, regardless of changes in outdoor conditions. In some bedrooms the Popmite predictions for the post-

intervention study are nil, suggesting the mite population in the mite bags could be completely eradicated after 6 weeks. However, in other bedrooms there is a smaller reduction in mite numbers: for example in Bedroom 1 there is only a 10% predicted reduction, based on the adjusted conditions. This suggests that - as already highlighted in the previous section - some households implemented the advice less effectively, or that the advice provided to them had a smaller potential for changes in hygrothermal conditions. As previously highlighted, the control Bedroom 6 experienced one of the highest reduction in adjusted RH levels, suggesting that their occupants might have changed their behaviours as a result of participating in the study.

Figure 11.6.1 shows the relationship between the difference from pre to post adjusted average bedroom RHs, and the reduction (ratio) in mite predictions corresponding to those changes in hygrothermal conditions. The graph shows that there are a few outliers, and that the two variables are not strongly correlated, due to those outliers[8]. The outliers are probably due to the strong threshold effects in Popmite (Chapter 9), where the impact of changes in RH (and temperature) depends on the hygrothermal conditions to which these changes are applied.

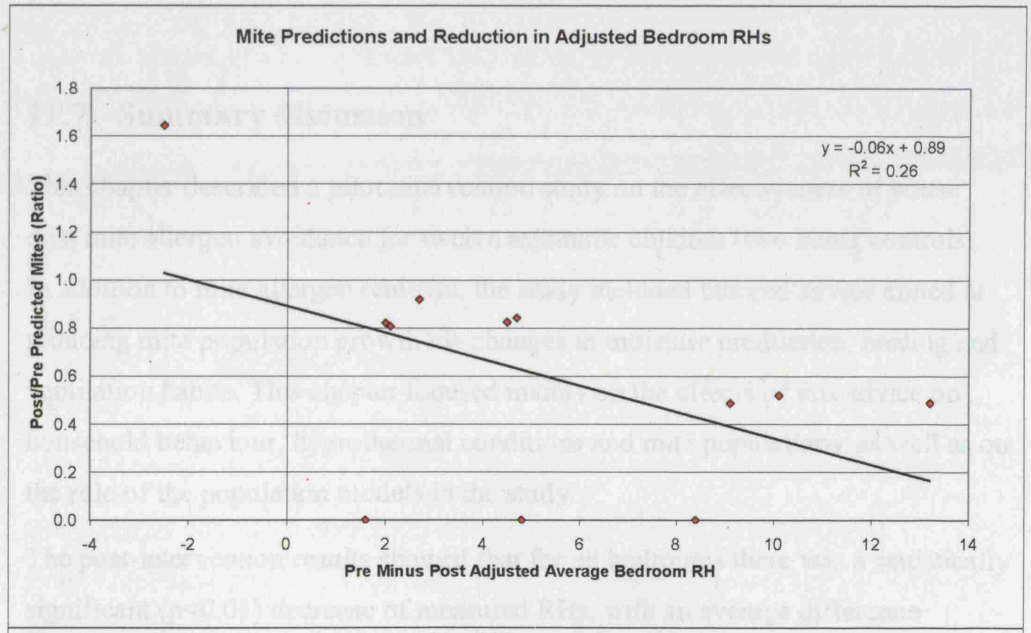


Fig 11.6.1 Relationship between the difference from pre to post adjusted average bedroom RHs, and the reduction (ratio) in mite predictions.

[8] If the outliers are removed from the data set, then the R-squared value becomes 0.76.

The presence of threshold effects means that the results based on *adjusted* hygrothermal conditions have to be taken with some caution, since *actual* conditions might impact on mite predictions with a different order of magnitude. This represents a challenge for any intervention study on the psychrometric control of house dust mites, if one wishes to examine the impact on mite populations of hygrothermal changes caused by the interventions alone, without the confounding factor of changes in outdoor conditions.

Another role of the combined hygrothermal population models in the intervention study would have been to model the dwelling in its pre-intervention conditions, and then simulate the impact (on mite populations) of changes in hygrothermal behaviours such as: increasing the thermostat setting, increasing the ventilation rates, etc. However, this time-consuming exercise did not match the tight schedule of the study. Furthermore, it is often rather difficult to accurately simulate specific *real* dwellings.

The next section is a summary discussion for the pilot intervention study and the role of the models in the study, as well as recommendations for larger scale studies.

11.7 Summary discussion

This chapter described a pilot intervention study on the effectiveness of house dust mite allergen avoidance for twelve asthmatic children (two being controls). In addition to mite allergen removal, the study included tailored advice aimed at reducing mite population growth via changes in moisture production, heating and ventilation habits. This chapter focused mainly on the effects of this advice on household behaviour, hygrothermal conditions and mite populations, as well as on the role of the population models in the study.

The post-intervention results showed that for all bedrooms there was a statistically significant ($p<0.01$) decrease of measured RHs, with an average difference between pre and post results of 12.1% (average excluding the control bedrooms). However, this RH reduction was partly due to changes in outdoor conditions. Once the effect of these changes was taken into account, the reduction from pre to post adjusted RHs was still statistically significant ($p<0.01$) but smaller, with an

average difference between pre and post adjusted bedroom RHs of 5.3%. The population modeling results indicated that during the pre-intervention period the mite populations were rather stable (average MPI $\cong$ 1), suggesting that even small hygrothermal changes could determine whether the population grows or declines. Therefore, even a 5% reduction in RH could lead to a decline in mite numbers, depending on the hygrothermal conditions to which such reduction is applied. Furthermore, this figure is likely to partly underestimate the RH reduction due to the implementation of the advice, since the adjustment procedure adopted in this chapter is more suitable for identifying changes in RH due to reductions in vapour pressure excess, rather than to changes in temperatures.

Some bedrooms experienced a lower than average reduction in RH, which suggests that some households had difficulties in implementing the advice, probably due to a combination of existing habits and adverse dwelling characteristics. For example, Household 1 was advised to increase ventilation rates, but their post-intervention VPX did not decrease, most probably because of their very airtight dwelling. This study shows that it can be difficult to determine to what extent occupants change their behaviour, particularly for ventilation habits. In order to assess hygrothermal changes, a combination of quantitative and qualitative data was used, which was complex to analyse due to: a) the lack of ventilation rate measurements[9]; b) difficulties in obtaining a coherent description of occupant habits, particularly for ventilation; c) the variability of such habits on a daily and seasonal basis; and d) the effect of changes in weather conditions. Adjusting for changes in outdoor conditions is particularly important when monitoring for short periods with varying external conditions. This is a limitation with almost all clinical-hygrothermal studies, since large sample sizes are usually required for clinical studies, but long-term hygrothermal monitoring is expensive. The adjustment method described in this chapter could be useful in this respect.

Since psychrometric control does not immediately affect HDM allergen reservoirs, these have to be removed in order to obtain any health improvement. However, this makes it difficult to assess the impact of RH reductions on mite populations, which are removed together with the allergen. Combined

[9] Air infiltration measurements were taken. However, ventilation rates can be affected by occupant behaviours.

hygrothermal population models and 'mite cages' can help to overcome this problem, as demonstrated in this study. However, in this study *adjusted* pre and post intervention hygrothermal conditions were utilised for predicting their effect on mite populations (caged mites). The adjusted conditions had to be adopted for two reasons: a) the mite bags could not be installed during the pre-intervention study, since this was too short; b) actual monitored results would have included the confounding factor of changes in outdoor conditions. The Popmite predictions based on the adjusted hygrothermal conditions showed that in all bedrooms (except in a bedroom acting as control) there was a reduction in the predicted number of mites in the post intervention period. This suggests that all dwellings implemented the advice to a sufficient level for mite growth reduction, regardless of changes in outdoor conditions. However, the reduction in adjusted RH levels was not strongly correlated with the predicted reduction in mite numbers from the pre to the post intervention periods. This is due to strong threshold effects in Popmite, where the impact of changes in RH (and temperature) on mite populations depends on the hygrothermal conditions to which these changes are applied. The presence of threshold effects means that the population predictions based on *adjusted* hygrothermal conditions have to be taken with some caution, since actual conditions might impact on mite predictions with a different order of magnitude. This represents a challenge for any intervention study on the psychrometric control of house dust mites, if one wishes to examine the impact on mite populations of hygrothermal changes caused by the interventions alone, without the confounding factor of changes in outdoor conditions.

The results of this study were promising with regard to the effect of HDM allergen reduction on rhinitis and asthma symptoms in children. However, the role of other confounding variables (e.g. sensitivity to other allergens) and the placebo effect were not properly controlled for in this study.

The following recommendations are made for future studies on the psychrometric control of house dust mites:

- Since hygrothermal changes do not immediately affect HDM allergen reservoirs, these have to be removed if any health benefits are to be gained. The placebo effect and the role of other allergens should be adequately addressed at the study design phase.

- It can be difficult to control/assess changes in hygrothermal behaviours. In order to facilitate this, ventilation rates and air infiltration should both be measured. The frequency of window opening and of extractor fans usage should ideally also be measured, both in the pre and the post intervention periods. Also, the selection of similar building types for the study would make the results more comparable, facilitating the assessment of the interventions' effectiveness.
- Any study attempting to modify indoor hygrothermal conditions has to develop an adequate method for adjusting against changes in weather conditions. Monitoring the dwellings for a whole season before the intervention (and a whole corresponding season after the intervention) would facilitate this process.
- A novel technique involving the use of 'mite cages' offers promising opportunities for assessing the potential impact of hygrothermal changes on mite populations over relatively short periods. For example, in those studies which include the removal of mite and allergen, as well as changes in hygrothermal conditions, it is not possible to assess the impact of such changes on mite populations – since the latter take some time to grow back.
- Hygrothermal and HDM population models are very useful to assess the effect of hygrothermal changes on mite infestations. They can be utilised, for example, to identify those dwellings most at risk of mite infestation and therefore help prioritise the interventions or allocate the case/control status. Furthermore, the population model Popmite might be utilised to assess the impact of pre and post intervention hygrothermal conditions on mite populations. If *adjusted* hygrothermal conditions are utilised in Popmite in order to exclude the effect of changes in outdoor conditions, then the results should be considered bearing in mind the strong threshold effects which characterise this population model.

Chapter 11: References

Arlian LG and Veselica MM, (1981), Effect of temperature on the equilibrium body water mass in the mite Dermatophagoides farinae, *Physiological Zoology*, 54(4): 393–399.

Coates D (Twenty Twenty Television), Producer of: *How to beat your kid's asthma*. Screened on Channel 4 on the 6 and 10 April 2006.

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

Howieson SG, Lawson A, McSharry C, Morris G, McKenzie E, Jackson J, (2003), Domestic ventilation rates, indoor humidity and dust mite allergens - Are our homes causing the asthma pandemic?, *Building Services Engineering, Research and Technology*, 24(3): 37-147.

Liddament MW, (2001), *AIVC Technical Note 53: Occupant Impact on Ventilation*, Brussels: AIVC.

Luczynska C, Sterne J, Bond J, Azima H, Burney P, (1998), Indoor factors associated with concentrations of house dust mite allergen, Der p 1, in a random sample of houses in Norwich UK, *Clinical and Experimental Allergy*, 28(10): 1201-1209.

Oreszczyn T and Pretlove SEC, (1999), Condensation Targeter II: Modelling surface relative humidity to predict mould growth in dwellings, *Building Services Engineering Research & Technology*, 20(3): 143-153.

Oreszczyn T, Hong S, Ridley I, Wilkinson P, The Warmfront Study Group, (2006), Determinants of winter indoor temperatures in low income households in England, *Energy and Buildings*, 38: 245-252.

Platts-Mills TAE and de Weck AL, (1989), Dust mite allergens and asthma - a worldwide problem, *Journal of Allergy and Clinical Immunology*, 83: 416–27.

Sanders C ed, (1996), *IEA Annex 24. Heat, Air and Moisture transfer through new and retrofitted insulated envelope parts. Final Report. Vol. 2, Task 2 Environmental conditions,* Belgium: Laboratorium Bouwfysica.

Sherman M, (1998), *The use of Blower Door Data,* Lawrence Berkeley Lab Report 35173, Berkeley: Lawrence Berkeley Laboratory.

Ucci M, Pretlove S, Biddulph P, Oreszczyn T, Wilkinson T, Crowther D, Scadding G, Hart B, Mumovic D, (2007), The psychrometric control of house dust mites: a pilot study, *Building Services Engineering and Technology Journal (BSERT)*, 28(4): 347-356.

Zock JP, Heinrich J, Jarvis D, Verlato G, Norbäck D, Plana E, Sunyer J, Chinn S, Olivieri M, Soon A, Villani S, Ponzio M, Dahlman-Hoglund A, Svanes C, Luczynska C and the Indoor Working Group of the European Community Respiratory Health Survey II (2006), Distribution and determinants of house dust mite allergens in Europe: The European Community Respiratory Health Survey II, *Journal of Allergy and Clinical Immunology*, 118(3): 628-690.

CHAPTER 12:

SCENARIOS MODELLING

CHAPTER 12: SCENARIOS MODELLING

12.1 Introduction

The ultimate goal of the models tested in this thesis is the identification of those building features and occupant behaviours, which are most effective for the prevention of house dust mite (HDM) growth in beds. The previous chapters discussed the validity of the combined hygrothermal population models, and their application in a pilot intervention study. This chapter discusses the use of the combined models for assessing the most effective psychrometric methods of HDM reduction in beds. In particular, the scenarios modelling focuses on the main building features and occupant behaviours which affect indoor hygrothermal conditions: insulation, airtightness, heating and ventilation patterns, and moisture production. The impact of outdoor conditions is also addressed, with a focus on different UK regions. The methodology utilised in this chapter involves considering the mite predictions for a base-case bed, in a base-case dwelling. Subsequently, one design (or occupancy) feature at a time is modified in the base-case dwelling. The mite predictions for the bed in each scenario are then compared with the base-case predictions. The predicted energy consumption of each scenario is also compared with the base-case consumption.

The next section (12.2) describes in more detail the methodology utilised for the scenarios modelling, while section 12.3 presents the results of the base-case model(s). Section 12.4 compares the base-case results against those from different scenarios. The chapter ends with a summary discussion (section 12.5).

12.2 Methodology and assumptions

This section illustrates the methodology and assumptions adopted for the scenarios modelling. This involved modelling a base-case dwelling and a base-case bed, and subsequently changing one building (or occupant behaviour) feature at a time, in order to assess the impact of changes in bedroom conditions on the bed's mite growth. Since the Popmite model predicted the fieldwork mite results (transient) more accurately than the MPI model (see Chapter 10), the transient set of hygrothermal population models was utilised for the scenarios modelling. In

particular, Lectus was adopted for the prediction of hygrothermal conditions in a bed, due to given room conditions. The Lectus predictions were then utilised in Popmite, in order to assess their impact on mite growth. The hourly room conditions required as inputs for the Lectus model were obtained by using the building simulation programme EnergyPlus 2 (US Department of Energy, 2007a), which has been validated against various standard methods (US Department of Energy, 2007b). EnergyPlus was selected since moisture adsorption/desorption of the internal surfaces can be modelled in this programme, with the effective moisture penetration depth model (Keresteciogl u *et al.*, 1990). The EnergyPlus materials library was utilised in order to define the EMPD properties of the indoor surfaces.

The base-case dwelling simulated in EnergyPlus was a 2-bedrooms mid-floor flat in the London area, with an occupancy of 4 people (2 adults and 2 children), 2 exposed walls, a floor area of 45 m^2 and a volume of 108 m^3 (Fig 12.1.2).

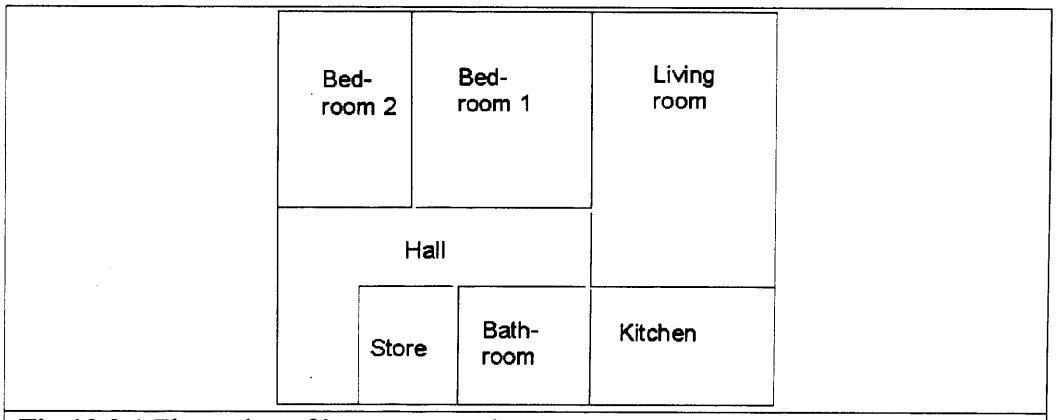


Fig 12.2.1 Floor plan of base-case study

This building type (flat) was selected since some authors have advocated that modern dwellings might be partly responsible for the rise in asthma prevalence in the last decades. This is because an increase in airtightness can result in higher levels of indoor moisture (Howieson *et al.*, 2003). Due to their small volumes and small exposed wall areas, flats can have rather low background infiltration rates. Furthermore, large concrete panel systems – often utilised in the construction of flats – have a low permeability, compared with other construction types (Stephen, 2000).

The base-case EnergyPlus model utilised in this chapter was originally created by Dr Ian Ridley (Bartlett School of Graduate Studies, UCL), for a Building Regulations research project on ventilation effectiveness (Palmer *et al.*, 2007). The author of this thesis made small changes to the original model, in order to obtain a base-case dwelling compliant with current building regulations (ODPM, 2006a)[1]. Subsequently, changes were made by the author to the base-case EnergyPlus model, in order to examine the impact of such changes on mite growth in beds. Table 12.1.1 summarises the main features of the base-case dwelling, whilst Table 12.1.2 summarises the changes made to the base-case model (i.e. the different scenarios).

Table 12.1.1 Details of the base-case dwelling modelled in EnergyPlus

Floor area	45 m^2
Volume	108 m^3
Envelope leakage (permeability)	10 $m^3 h^{-1} m^{-2}$ at 50 Pa
Envelope insulation (U-value)	**Walls**: 0.35 W $m^{-2} K^{-1}$ **Windows**: 2.2 W $m^{-2} K^{-1}$
Trickle vents (equivalent areas)	**Bedroom 1**: 10000 m^2 **Living Room & Kitchen**: 12500 m^2 in each room **Bedroom 2 & Bathroom**: 7500 m^2 in each room **Total**: 50000 m^2
Extract fan	**Kitchen**: 60 l/s (intermittent use) **Bathroom**: 15 l/s (intermittent use)
Heating system	**Thermostat set point** (living room): 20 °C **Heating season**: 1st October to 31st of May **Size of electric heaters**: 2 kW in each room **Hours heating per day**: 10 hours at weekdays, 17 hours at weekends.
Window opening	10% open (intermittent use)
Moisture input	Equivalent to 6 kg/day (moist occupancy*)
Outdoor climate	London (EnergyPlus weather file: Present-kew.epw)

*BSI, 2002

[1] The original model from Dr Ridley had lower permeability and U-values than required by current building regulations.

Table 12.2.2 Changes made to the EnergyPlus base-case model.

	Variable changed	Base-case value	Option 1	Option 2
1	Permeability at 50 Pa	10 $m^3 h^{-1} m^{-2}$	20 $m^3 h^{-1} m^{-2}$	3 $m^3 h^{-1} m^{-2}$
2	Walls U-value	0.35 $W m^{-2} K^{-1}$	1.6 $W m^{-2} K^{-1}$	0.25 $W m^{-2} K^{-1}$
3	Window Opening (time)	10% open, intermittent times	As base case, but also open all night in bedrooms	Always closed in bedrooms
4	Extract fan (time)	Intermittent use	Additional 2 hours usage from base-case	Fans never used
5	Thermostat setting	20 °C	22 °C	18 °C
6	Heating hours	10 hours at weekdays, 17 hours at weekends	12 hours at weekdays, 19 hours at weekends	8 hours at weekdays, 15 hours at weekends
7	Total daily moisture input	6 kg/day (Moist occupancy*)	14 kg/day (Wet occupancy*)	5 kg/day (Dry occupancy*)

* BSI, 2002

Table 12.2.2 shows that for each variable modified in the base-case dwelling, two options were considered (respectively with a higher and a lower value than base-case), so that a *range* of input variables could be examined. In each case, the values for option 1 and 2 were chosen as representative of ranges found in real dwellings. For example, in the "moisture input" case, option 1 corresponds to "wet occupancy", while option 2 corresponds to "dry occupancy" (following the definition given in: BSI, 2002).

In addition to the options illustrated in Table 12.2.2, a balanced mechanical supply and extract ventilation system with heat recovery (MVHR) was also applied to the base-case flat, in order to assess its impact on mite growth in beds. The initial MVHR model had also been created by Dr Ian Ridley, for the Building Regulations research project on ventilation effectiveness (Palmer *et al.*, 2007). Three MVHR options were tested in this chapter: 1) option one, with ventilation rates compliant with current building regulations requirements (ODPM, 2006b); 2) option two, with greater ventilation rates (40% greater than option one); 3) option three, based on option two but with a bypass system for the heat recovery in the non-heating season. The details of the MVHR options are provided in Table 12.2.3.

Table 12.2.3 Details of the mechanical ventilation with heat recovery systems, added to the base-case dwelling.

Sensible heat recovery	90%
Whole building ventilation rate	Option 1: 14.5 l/s
Whole building ventilation rate	Option 2: 21 l/s (same as option 3)
Continuous mechanical extract	**Kitchen, *Case 1***: 9.0 l/s (12 l/s boost); **Kitchen, *Case 2***: 13 l/s (18 l/s boost); **Bathroom, *Case 1***: 5.5 l/s (8 l/s boost); **Bathroom, *Case 2***: 8 l/s (11 l/s boost);
Continuous mechanical supply	**Living room, *Case 1***: 6.5 l/s (9 l/s boost); **Living room, *Case 2***: 9 l/s (12.5 l/s boost); **Bedroom 1, *Case 1***: 4 l/s (6 l/s boost); **Bedroom 1, *Case 2***: 6 l/s (8.3 l/s boost); **Bedroom 2, *Case 1***: 4 l/s (6 l/s boost); **Bedroom 2, *Case 2***: 6 l/s (8.3 l/s boost);
System boost period, week day	07:00 to 08:30, 18:00 to 20:30, 21:30 to 22:00
System boost period, week day	08:00 to 09:30, 12:00 to 12:30, 18:00 to 20:30, 21:30 to 22:00
Option 3	Same as Option 2, but no heat recovery in the non-heating season (1st June to 31st September)

Outdoor conditions for the London area had been utilised for the base-case dwelling (see Table 12.1.1). In order to assess the impact of changes in outdoor conditions, different weather files were utilised with the base-case dwelling, representing the following UK regions: Scotland, North of England, Wales. The weather files for Scotland (Aberdeen) and North of England (Doncaster) were downloaded from the EnergyPlus website (US Department of Energy, 2007c), while the weather file for Wales (Aberporth) was obtained from Meteonorm 4.0 (Meteotest, 1999). These weather files were selected since they were accessible to the author, and were utilised in order to give an indication of the likely effect of different outdoor conditions on mite populations. It should be mentioned however that other locations within the UK might be even more favourable to mite growth than those tested in this Chapter.

The energy consumption of each option considered in EnergyPlus was also compared with the base-case energy consumption.

The hourly hygrothermal conditions (1 year) predicted by EnergyPlus for each of the options described so far were utilised as inputs in the Lectus model. In particular, the EnergyPlus results from bedroom 2 were utilised, since this bedroom had a greater occupancy per floor area than bedroom 1 (i.e. greater moisture). The mattress modelled in Lectus was a 15 cm thick homogenous single mattress, with material properties equivalent to foam. These properties were

collated from a number of sources (Svennberg *et al.*, 2005; Trechsel ed., 1994; Pretlove *et al.*, 2005) and are summarised in Table 12.2.4, together with the other input variables utilised in the base-case Lectus model. The sensitivity analysis for the Lectus model had shown that changes in mattress properties had no dramatic impact on the model's predictions, when compared to changes in room and boundary conditions (Chapter 9). Therefore, the mattress properties are not so crucial, compared with other input variables. For this reason, it was also decided not to include changes in mattress properties in the scenarios modelling. Figure 12.2.2 shows the Lectus boundary conditions for the top mattress surface, when the bed is occupied.

Table 12.2.4 Main inputs for the Lectus model (base-case)

Input Parameter	
Density	36 Kg/m^3
Thermal Conductivity	0.06 W/mK
Heat Capacity	850 J/kgK
Vapour Permeability	2.33E-12 kg/msPa
Moisture Capacity	2.00E-05 kg/kgPa
Thickness	0.15 m
Time in Bed	8 hours
Half Life*	30 minutes
Boundary Conditions	(see Fig 12.2.1)
Calculations time-step	100 seconds
Room Conditions	(from EnergyPlus)

*Time it takes for the difference between the mattress top surface conditions and the room conditions to be halved, once the bed is vacated.

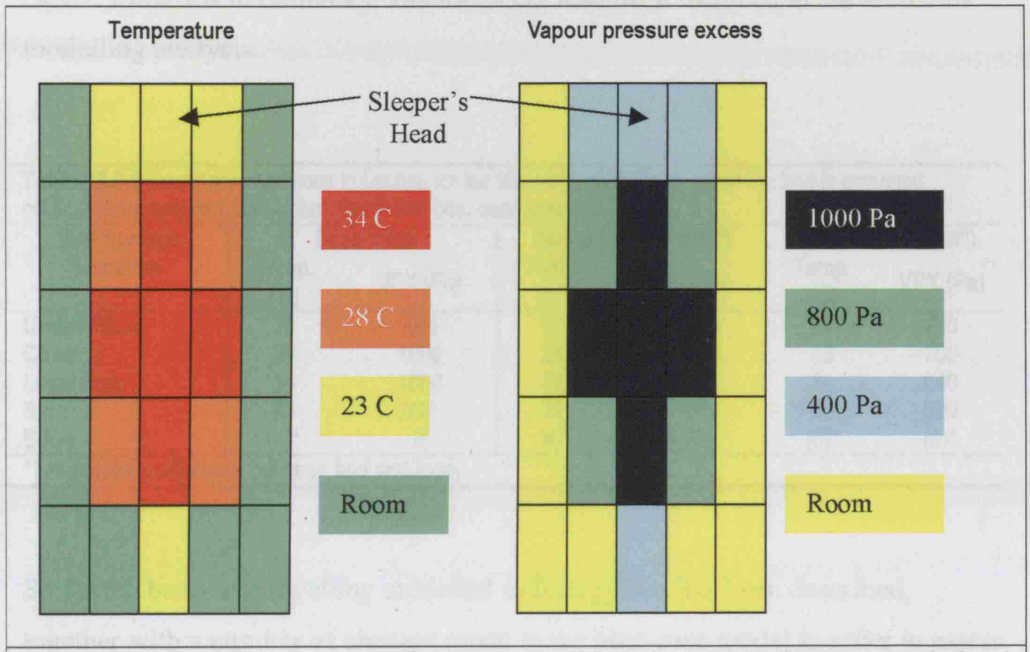


Fig. 12.2.2 Top of the mattress boundary conditions in Lectus, for an occupied mattress (base-case). When the bed is unoccupied, the top mattress surface has the same conditions as the room. The other mattress surfaces are always assumed in equilibrium with room conditions.

The mattress simulated in Lectus was divided into 4 layers, whose thickness was (from the top to bottom of the mattress): 2.5 cm, 2.5 cm, 5 cm, 5 cm. The first two layers were thinner than the other two, in order to test the finding based on preliminary measurements that the most favourable location for mite growth in a homogeneous bed is at 1-2 cm below the top mattress surface (Ridley *et al.*, submitted). In the Lectus model, each mattress layer was divided into 5x5 cells, which replicates the division of the top mattress surface as assumed in the Lectus boundary conditions (see Fig 12.2.2). This corresponded to a total of 100 cells. It was found that doubling the number of cells for each mattress layer produced predictions within +/- 1% of the predictions for the 100 cells case.

The comparison of the Lectus boundary conditions with fieldwork data had shown that although *on average* the boundary conditions assumed in Lectus are sufficiently representative of fieldwork data, a *range* of conditions are likely to occur in reality (Chapter 5). This range of boundary conditions were summarised in Chapter 5 and are replicated in Table 12.2.5. Since Lectus is sensitive to

changes in boundary conditions (Chapter 9), the impact of "best case"[2] and "worst case"[3] scenarios in boundary conditions has also been included in the scenarios modelling analysis.

Table 12.2.5 Boundary conditions in Lectus, for the top mattress surface, when the bed is occupied: original assumptions (base-case), worst and best case scenarios.

Bed Surface Location	Base-case		Scenario 2 ("worst")		Scenario 3 ("best")	
	Temp. (°C)	VPX (Pa)	Temp. (°C)	VPX (Pa)	Temp. (°C)	VPX (Pa)
Under Pillow	23	400	24	550	22	200
Chest	34	1000	35	1300	33	700
Legs/Feet	34	1000	35	1300	31	640
Side	28	800	29	1080	26	520
Edge	(0)*	0	(4)*	100	(0)*	(0)*

*Temperature difference between bed and room.

So far the base-case dwelling modelled in EnergyPlus has been described, together with a number of changes made to the base-case model in order to assess their impact on mite growth in beds (Table 12.2.1-12.2.3). For each of these options, the EnergyPlus predictions for bedroom 2 were utilised as input in the Lectus model, whose other base-case inputs have also been illustrated (Table 12.2.4). This resulted in 100 Lectus output files for each option (hourly hygrothermal conditions, 1 year), corresponding to the 100 mattress cells. The base-case Lectus files were then utilised as input in the Popmite model, whose final output is the final mite population after one year, in each cell.

It should be mentioned that the Lectus files utilised in Popmite were modified, so that the starting date in each file was the 1st of August. This is because studies have shown that mite levels are higher in late summer-early autumn (Crowther *et al.*, 2006).

Popmite requires as input the starting population in each mattress cell. However, currently there is insufficient information on the exact number and distribution of live mites in a mattress, mostly due to difficulties associated with sampling live mites (see Chapter 2). Consequently, the starting population in each modelled mattress cell was assumed as 20 adult mites (spread of all ages). This figure was selected since it was the starting population in the fieldwork study (i.e. mite bags).

[2] Lower temperature and lower vapour pressure excess (worse for mites)
[3] Higher temperature and higher vapour pressure excess (better for mites)

It should also be pointed out that mite movement across the mattress cells is currently not modelled in Popmite/Lectus, nor any restrictions are included in Popmite on food or space availability. This is because further research is required on these parameters, before they can be incorporated in Popmite. However, the purpose of the scenarios modelling described in this chapter is to identify those variables which have the potential to *increase* or *decrease* mite growth in a bed, compared with a base-case dwelling. The scenarios modelling does not aim to establish the exact *number* of mites in a bed, due to the various scenarios. Consequently, the *size* of the starting population in each cell is, to a certain extent, irrelevant. In some circumstances, however, the initial population size might be important: if the initial population is large enough, it may survive unfavourable conditions, in comparison with a small starting population. On the other hand, if the initial population is extremely large, this might lead to some aberrations: in reality there will be food and space restrictions, which are currently not modelled in Popmite. Conversely, if the starting population is too small, then it might be too vulnerable to dry spells. In these circumstances, the effect of certain scenarios *might* be overlooked. The issue of the initial population size is further addressed later in the chapter. However, it should also be mentioned that, despite the uncertainties on the starting population and although the lack of food and space restrictions in Popmite are unrealistic, Popmite is based on state of the art knowledge on house dust mites. Furthermore, in most circumstances the aforementioned uncertainties would still enable to establish whether the population will *increase* or *decrease* from a base-case scenario, which is the aim of this chapter. Finally, it should also be highlighted that although the EnergyPlus model has been extensively validated (US Department of Energy, 2007b), building simulation models are more suitable at modelling the impact of changes (from a base-case), than absolute values (same as population models). For example, the impact of occupant behaviour is often difficult to model. However, in this study the EnergyPlus model is utilised to *compare* results against a base-case.

The next section illustrates the base-case results. Table 12.2.6 summarises *all* the variables considered for scenarios modelling.

Table 12.2.6 Summary of *all* variables changed in the base-case model.	
1	Permeability at 50 Pa
2	Walls U-value
3	Window Opening (time)
4	Extract fan (time)
5	Thermostat setting
6	Heating hours
7	Total daily moisture input
8	Mechanical ventilation with heat recovery
9	Boundary conditions (Lectus)
10	UK climatic regions

12.3 Base-case results

This section illustrates the results from the base-case model(s), whose details were provided in the previous section.

Table 12.3.1 shows a summary of the hygrothermal conditions predicted by EnergyPlus for bedroom 2 of the base-case dwelling. The total predicted energy consumption of the base-case flat was 4278 kWh/year.

Table 12.3.1 Average and standard deviation of the temperature and RH predicted by EnergyPlus for bedroom 2 of the base-case flat

	Heating Season*	Non-Heating Season#
Bedroom 2: Temp., Average (°C)	18.1	20.3
Bedroom 2: Temp., St. Dev. (°C)	2.4	2.0
Bedroom 2: RH, Average (%)	52.1	68.7
Bedroom 2: RH, St. Dev. (%)	11.8	8.2
Outdoor: Temp., Average (°C)	8.0	16.7
Outdoor: Temp., St. Dev. (°C)	4.1	3.5
Outdoor: RH, Average (%)	79.9	72.5
Outdoor: RH, St. Dev. (%)	10.6	11.2

* 1st Oct. to 31st May; # 1st June to 30th Sept.

The hygrothermal conditions predicted by EnergyPlus for bedroom 2 were utilised in Lectus, whose output files were then adopted in Popmite. This enabled to establish the final number of mites in each mattress cell after 12 months, based on a starting population of 20 adult mites for each cell. Figure 12.3.1 shows the base-case predicted final total number of mites (incl. eggs), for each mattress cell, by mattress layer. As previously mentioned, the mattress was divided into 4 layers, each with a grid of 5x5 cells. Figure 12.3.1 also shows the mite predictions for the top mattress surface, whose hygrothermal conditions are the same as the room when the bed is unoccupied, while when the bed is occupied the boundary

conditions are illustrated in Figure 12.2.1. The other 5 surfaces of the mattress are always assumed in equilibrium with room conditions and therefore have the same mite numbers as the perimeter cells of the mattress's top surface - which are in equilibrium with room conditions at all times, see Figure 12.2.1.

Figure 12.3.1 Base-case: schematic representation of the predicted final number of mites (total, including eggs) for each mattress layer, after 12 months. Starting population in each cell: 20 adult mites.

Mattress Plan: Top Surface (Boundary)

3,574	3,574	3,574	3,574	3,574
0	0	0	0	3574
Head: 0	Chest: 0	Groin: 0	Legs: 0	Feet: 0
0	0	0	0	3,574
3,574	3,574	3,574	3,574	3,574

Key to Colours

Number of Mites (total, incl. eggs)	Code
<3	
Approx. 400	
Approx. 1,000	
Approx. 2,000	
Approx. 3,000-4,000	
Approx. 5,000	
Approx. 10,000	
Approx. 13,000-20,000-	
Approx. 50,000	
Approx. 600,000-800,000	
Approx. 2.4mill.-2.9mill.	

Mattress Plan: Middle of 1st Layer (from top)

2,930	3,043	3,095	3,042	2,854
4,882	157,318	2,384,023	155,157	2,985
Head: 4,853	Chest: 2,516,252	Groin: 2,858,465	Legs: 2,503,486	Feet: 5,237
4,882	157,318	2,384,023	155,157	2,985
2,930	3,043	3,095	3,042	2,854

Mattress Plan: Middle of 2nd Layer (from top)

1,765	1,795	1,886	1,789	1,759
2,034	54,409	632,523	52,844	1,686
Head: 1,985	Chest: 658,633	Groin: 789,137	Legs: 655,473	Feet: 1,481
2,034	54,409	632,523	52,844	1,686
1,765	1,795	1,886	1,789	1,759

Mattress Plan: Middle of 3rd Layer (from top)

1,128	1,160	1,224	1,151	1,107
1	355	9,296	374	1,025
Head: 3	Chest: 13,471	Groin: 19,830	Legs: 13,454	Feet: 0
1	355	9,296	374	1,025
1,128	1,160	1,224	1,151	1,107

Mattress Plan: Middle of 4th Layer (from top)

2,039	1,987	2,096	1,984	1,975
1,775	2,195	1,982	2,365	1,943
Head: 1,737	Chest: 1,786	Groin: 1,744	Legs: 1,921	Feet: 1,728
1,775	2,195	1,982	2,365	1,943
2,039	1,987	2,096	1,984	1,975

Figure 12.3.1 shows that in the middle of the first layer (1.25 cm from the top mattress surface), the number of predicted final mites is greatest, particularly in the area – shaped liked a cross - corresponding to the chest, groin and legs. On the other hand, in the same area but on the *top* mattress surface, all the mites are dead. This is because - as already shown by previous studies (Cunningham *et al.*, 2004; Ridley *et al.*, submitted) - when the bed is occupied the additional moisture due to the sleeper is counteracted by the occupant's high body temperature, which produces low RHs. As a result, conditions on the top mattress surface (under the body) are too dry for the mites to survive. This was confirmed by the fieldwork study, where nearly all the mites in the mite bags positioned on the top mattress surface under the chest area died after 6 weeks. On the other hand, below the mattress top surface the excess moisture from the body does result in greater RHs than room conditions, which facilitates mite growth. This is particularly true for the first mattress layer, which appears the most favourable location for mites. This is unfortunate, since this location is also likely to have plenty of food (skin scales), from the bed's occupier.

It should be noted that the highest number of mites per mattress cell predicted by Popmite is rather high: nearly 2.9 millions (layer 1, groin area). This figure should be taken with some caution, since Popmite over-predicts fieldwork results, particularly at high RHs (Chapter 7). Furthermore, Wilkinson *et al.* (2002) studied the impact of starting population, food and space availability on the carrying capacity (K) of a mattress on populations of house dust mites (*Dermatophagoides Pteronyssinus*). They found that K is affected by both food quantity/quality and space. Also, they found that the initial population affected final mite numbers and that mite migration increased significantly, once certain threshold levels were reached. However, as previously mentioned Popmite does not currently include the effect of food/space constrains or mite movement across cells. Since the mite distribution in a mattress *is* affected by food and space availability, as well as by population numbers/movement (Wilkinson *et al.*, 2002), the mite distribution predicted by Popmite for the base-case mattress may be different in a real bed. However, until further detailed information becomes available on the other factors affecting such distribution (other than hygrothermal conditions)[4], Popmite still

[4] I.e. food, space, movement, typical population sizes per volume

represents the best available model for assessing the impact of transient hygrothermal conditions on HDM populations.

As already mentioned in the previous section, the Lectus files were utilised in Popmite so that the hygrothermal conditions would start from the 1st of August (for 12 months). Late summer/early autumn is the most favourable period for mite growth in temperate climates, whilst the heating season is considered the least favourable for mites. This is due to the low absolute moisture content of outdoor air resulting in lower indoor RHs, once the outdoor air is sufficiently heated (Crowther *et al.*, 2006). Therefore, it was decided to assess whether a significant change in the base-case mite predictions would be obtained, if the starting date for the hygrothermal input conditions was the 1st of November (heating season), as opposed to the 1st of August.

Figure 12.3.2 shows the impact of two simulation start dates (1st Nov and 1st Aug) on predicted mite numbers after the 12 months, for the mattress cell with the greatest predicted final numbers (layer 1, groin area). The graph shows that the predicted final number of mites is higher, if the starting input date is the 1st of August, rather than the 1st of November. This has to be highlighted to future non-expert users of the population model. It should also be highlighted that in reality the mattress mite population will be partly replenished through mite migration from other sources.

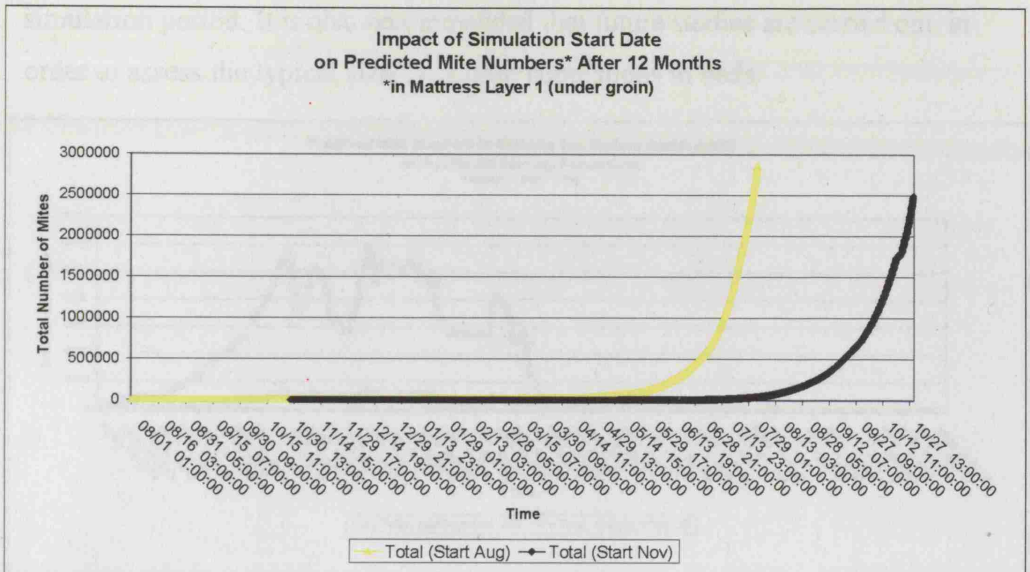


Fig 12.3.2 Impact of simulation start date on predicted mite numbers over 12 months.

The previous graph showed the mite results for the mattress cell with the greatest predicted mite numbers after 12 months. It might be interesting to consider a mattress cell with nil final predicted mites, and assess whether a greater initial population (e.g. 200 adult mites) might change the predictions, so that some live mites are left after 12 months. This is because in the Popmite model 10% of the mite population dies every hour, if the conditions are unfavourable. Therefore, it is theoretically possible that in some cases a sufficiently large initial population is not completely eradicated under unfavourable conditions. Figure 12.3.3 compares the total number of mites predicted for an initial population of 20 mites, and for an initial population of 200 mites (start date: 1st Aug), for a cell whose base-case predictions were nil. As expected, the graph shows that if the starting population is higher, its peak is higher than with a lower starting population, when conditions are favourable. However, the final population after 12 months is still nil - although it takes longer for all the mites to die, if the starting population is 10 times higher than the base-case. This means that if hygrothermal conditions are unfavourable for a sufficient amount of time, the size of the initial start population is not crucial in determining whether mites are all completely eradicated. However, in future it is still advisable to test whether the chosen population size can affect the results, in relation to the length which has been chosen for the

simulation period. It is also recommended that future studies are carried out, in order to assess the typical size(s) of mite populations in beds.

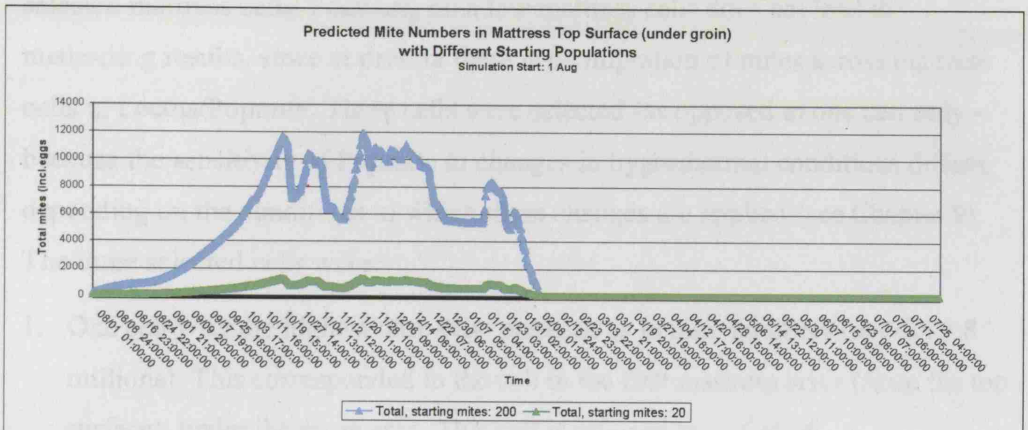


Fig 12.3.3 Predicted total mite numbers (incl. eggs) for the top surface mattress cell (under groin), with a starting population of: 20 mites and 200 mites.

This section described the results of the base-case dwelling/mattress. The results showed that the first layer from the top mattress surface (1.25 cm from top) is the most favourable to mite growth, particularly in the areas correspondent to the sleeper's groin, chest and legs. For the same areas, the final number of live mites predicted on the *top surface* of the bed was nil. The results also showed that the initial number of mites and the initial input hygrothermal conditions (late summer versus heating season) have an impact on the model's predictions. Since Popmite was proved to over-predict fieldwork results, particularly at higher RHs, some of the results described so far on the distribution of mites in the base-case mattress should be taken with caution. Furthermore, such results might be different, if more information on the effect of food/space constraints and on mite movement was available and could be incorporated into Popmite.

The following section illustrates the results from the scenarios modelling.

12.4 Scenarios modelling results

The previous section described the mite predictions for the base dwelling/bed. This section discusses the results for the various scenarios illustrates in section 12.2.

Since the computational time for Popmite predictions of all the 100 mattress cells was rather long (approx. 3 hours), it was decided to focus the analysis on three selected mattress cells. Focusing on a few mattress cells does not lead to misleading results, since at present there is no migration of mites across mattress cells in Lectus/Popmite. Three cells were selected -as opposed to one cell only - because the sensitivity of Popmite to changes in hygrothermal conditions differs, depending on the conditions to which these changes are applied (see Chapter 9). The three selected cells were:

1. One cell with the highest base-case predictions in mite numbers (approx 2.8 millions). This corresponded to the cell in the first mattress layer (from the top surface), under the groin area. This cell is referred to as *Cell A*.
2. One cell with low base-case predictions in mite numbers (355 mites)[5]. This corresponded to the cell in the mattress third layer (from the top surface), next to the chest area. This cell is referred to as *Cell B*.
3. A cell with the same hygrothermal conditions as the room, which had a base-case predicted mite numbers of 3574. This cell is referred to as *Cell C*.

Table 12.4.1 summarises the mite predictions for the base-case cells and the energy consumption of the base-case flat, whilst Table 12.4.2 shows the mite and energy consumption predictions for all the scenarios detailed in section 12.2 (except changes in outdoor conditions). The results in Table 12.4.2 are given in terms of *ratio* between the predictions for a specific scenario, and the predictions for the base-case. The average temperature and RH values, predicted for each of the three cells in each scenario, are provided in Figure 12.4.1 and 12.4.2. It should be mentioned that EnergyPlus does not accurately model moisture movement through the building fabric, which will partly affect indoor RHs and energy consumption values.

[5] Those few cells with very small (<10) or nil predictions were not considered for the scenarios modelling, since it was assumed that in these cells changes in hygrothermal conditions might still result in predictions of nil mite numbers.

Table 12.4.1 Summary results for the base-case

	Mite Predictions After 12 Months (total number of mites, incl. eggs)			Energy Consumption (kWh/year)
	Cell A	Cell B	Cell C	
Base-Case	2,858,465	355	3,574	4,278

Table 12.4.2 Scenarios modelling results

	Mite Predictions*			Energy Cons.*
Scenarios	Cell A	Cell B	Cell C	
1) U-value: 1.6 W/m²K	0.65	8.77	0.62	1.27
2) U-value: 0.25 W/m²K	1.10	1.03	1.10	0.97
3) Permeability: 20 m³/m²h	0.08	0.38	0.20	1.58
4) Permeability: 3 m³/m²h	269.05	2538.55	31.73	0.55
5) Windows open all night	0.75	0.91	0.75	1.02
6) Windows closed	1.03	1.16	0.99	1.00
7) Extract fan, longer use	0.45	0.35	0.58	1.07
8) No extract fan	5.49	54.37	2.43	0.89
9) Thermostat: 22 °C	0.03	0.00	0.88	1.20
10) Thermostat: 18 °C	5.41	15.50	0.41	0.80
11) Heating period: plus 2 hours	0.94	0.08	1.05	1.05
12) Heating period: minus 2 hours	5.59	54.24	0.82	0.94
13) Moisture: 14 kg/day	7.08	55.61	1.83	1.00
14) Moisture: 5 kg/day	0.02	0.00	0.03	1.00
15) MVHR, option 1	159.67	369.69	25.37	0.31
16) MVHR, option 2	20.43	0.00	0.07	0.33
17) MVHR, option 3	7.32	42.60	1.32	0.51
18) U-value 0.25 W/m²K and permeability 3 m³/m²h	312.63	3032.87	38.78	0.52
19) Boundary conditions: best case	0.10	0.73	(1.0)#	(1.0)#
20) Boundary conditions: worst case	10.11	45.91	(1.0)#	(1.0)#

* Ratio with Base-Case; #No changes expected

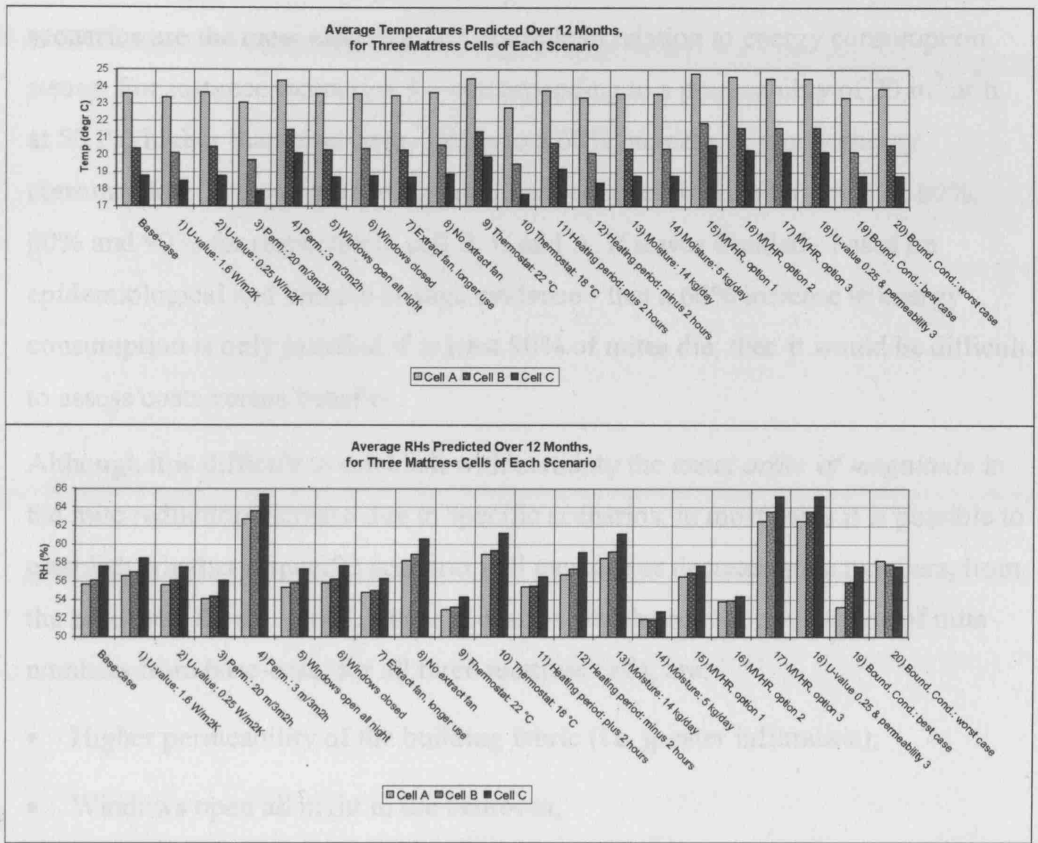


Fig 12.4.1 and 12.4.2 Average hygrothermal conditions (over 12 months) for the three mattress cells, in each scenario.

The results in Table 12.4.2 show that the changes in scenarios affect the 3 mattress cells with different orders of magnitude. For example, in scenario 4 (permeability of 3 m^3/m^2h at 50 Pa, lower than base-case), the ratio of mite predictions with base-case predictions is: 269, 2538.5 and 31.7, respectively for cell A, B and C. Furthermore, in some cases a certain scenario leads to a *reduction* in predicted mite numbers for one cell, but to an *increase* in predicted mite numbers for another cell. For example, in scenario 1 (corresponding to a U-value of 1.6 W/m^2K, higher than base-case) the ratio of mite predictions with base-case is: 0.6, 8.8 and 0.6 respectively for cell A, B and C. This confirms that, due to threshold effects, the sensitivity of Popmite to changes in hygrothermal conditions differs, depending on the conditions to which these changes are applied (see Chapter 9).

Since the impact of some scenarios on the 3 mattress cells results on different orders of magnitudes in the predictions, it can be difficult to assess whether such scenarios are the most effective, for example in relation to energy consumption issues. For instance, scenarios 3 – corresponding to a permeability of 20 m^3/m^2h at 50 Pa, higher than base-case – leads to a 60% increase in yearly energy consumption. The corresponding predicted reduction in mite numbers is: 60%, 80% and 90% for respectively cell B, C and A. If it was decided - based on epidemiological and climate change evidence - that a 60% increase in energy consumption is only justified if at least 90% of mites die, then it would be difficult to assess costs versus benefits.

Although it is difficult to establish with certainty the *exact order of magnitude* in the mite reduction/increase due to specific scenarios, in most cases it is possible to establish whether a specific scenario will increase or decrease mite numbers, from the base-case dwelling/bed. Those scenarios which produce a *reduction* of mite numbers from base-case, for all three mattress cells, are:

- Higher permeability of the building fabric (i.e. greater infiltration);
- Windows open all night in the bedroom;
- Longer use of extract fan;
- Higher thermostat setting;
- Lower moisture production.

Those scenarios which produce an *increase* of mite numbers from base-case, for all three mattress cells, are:

- Lower U-value;
- Lower permeability;
- Window closed;
- No extract fan;
- Higher moisture production;
- Lower U-value and permeability.

It is perhaps unexpected that a lower U-value should slightly *increase* the number of mites (case 2 in Table 12.4.2). Higher indoor temperatures should lead to smaller RHs. However, higher temperatures also reduce development times in mites, and Popmite is at least as sensitive to changes in temperature, as to changes in RH (Chapter 9).

The sensitivity analysis had shown that Popmite is a complex model, with strong threshold effects, affected by both temperature and RH changes (as well as eating rates), often in unpredictable ways. For example, a 10% increase in RH does not lead to a fixed increase in mite numbers, since this figure depends on the initial hygrothermal conditions to which the 10% increase in RH is applied. Furthermore, an increase in temperature might lead to a *decrease* or *increase* in mite predictions, depending to the hygrothermal conditions to which such change is applied to. This is probably the reason why some scenarios considered in this chapter result in a *reduction* of mite numbers (from base-case) in one of the mattress cells, and to an *increase* in another mattress cell (see cases 1, 10, 11, 12, 16 of Table 12.4.2).

The complexity in Popmite predictions is also highlighted by the three MVHR options (Table 12.4.2, case: 15, 16, 17). MVHR Option 1 determines an increase in mite predictions, compared with the base-case. Option 2 (greater ventilation rates than Option 1) results in a reduction of mite predictions for two of the three selected mattress cells. On the other hand, Option 3 (as Option 2, but heat recovery switched off in summer) increases mite predictions, but to a lesser extent than Option 1. In view of these results, it is perhaps hardly surprising that some field trials of HDM control via MVHR have been not conclusive. Figure 12.4.3 and 12.4.4 show the temperature and RH distributions over 12 months, for the three MVHR options and for the base-case. The graphs show that Option 1 has greater temperature and RH values than the base-case and than Option 2. On the other hand, Option 3 has a more right-skewed distribution in RHs than Option 1, but no temperatures in the 25-30 °C region. Although MVHR is often advocated as a method for moisture reduction, the use of purge ventilation with higher ventilation rates at targeted times (i.e. base-case: extract fan) can be more effective at removing moisture than smaller but continuous ventilation, as in the MVHR Option 1 case (Palmer *et al.*, 2007). Although the increase in temperature

resulting from the heat recovery in the MVHR system should result in a further RH reduction, such temperature increase might also result in more favourable conditions for mites.

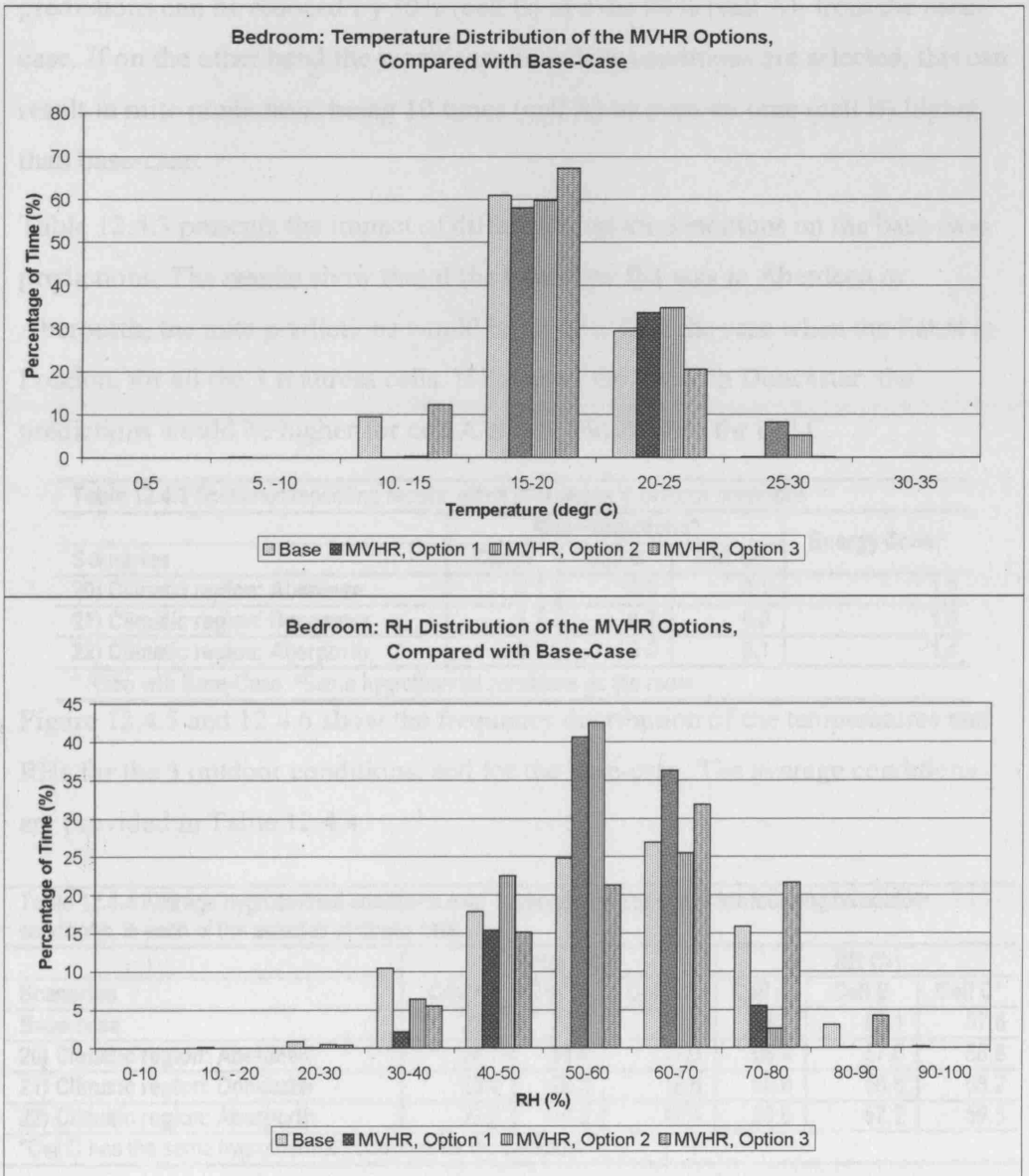


Fig 12.4.3 and 12.4.4 Temperature and RH distribution over 12 months for the three MVHR options and the base-case.

Table 12.4.2 also showed the results of two alternative scenarios for the mattress boundary conditions (case 19 and 20). The comparison of the Lectus boundary conditions with fieldwork data had revealed that although *on average* the boundary conditions assumed in Lectus are sufficiently representative of

fieldwork data, a *range* of conditions are likely to occur in reality (Chapter 5), representing a "best case"[6] and "worst case"[7] scenarios. The scenarios modelling results show that if best case boundary conditions are selected, the mite predictions can be reduced by 30% (cell B) or even 90% (cell A), from the base-case. If on the other hand the worst case boundary conditions are selected, this can result in mite predictions being 10 times (cell A) or even 46 time (cell B) higher than base-case.

Table 12.4.3 presents the impact of different outdoor conditions on the base-case predictions. The results show that if the base-case flat was in Aberdeen or Aberporth, the mite predictions would be smaller than the case when the flat is in London, for all the 3 mattress cells. If however the flat is in Doncaster, the predictions would be higher for cell A and B, but smaller for cell C.

Table 12.4.3 Scenarios modelling results: effect of changes in outdoor conditions

	Mite Predictions*			Energy Cons.*
Scenarios	**Cell A**	**Cell B**	**Cell C#**	
20) Climatic region: Aberdeen	0.1	0.6	0.1	1.2
21) Climatic region: Doncaster	1.2	4.7	0.6	1.0
22) Climatic region: Aberporth	0.1	0.0	0.1	1.4

* Ratio with Base-Case; #Same hygrothermal conditions as the room

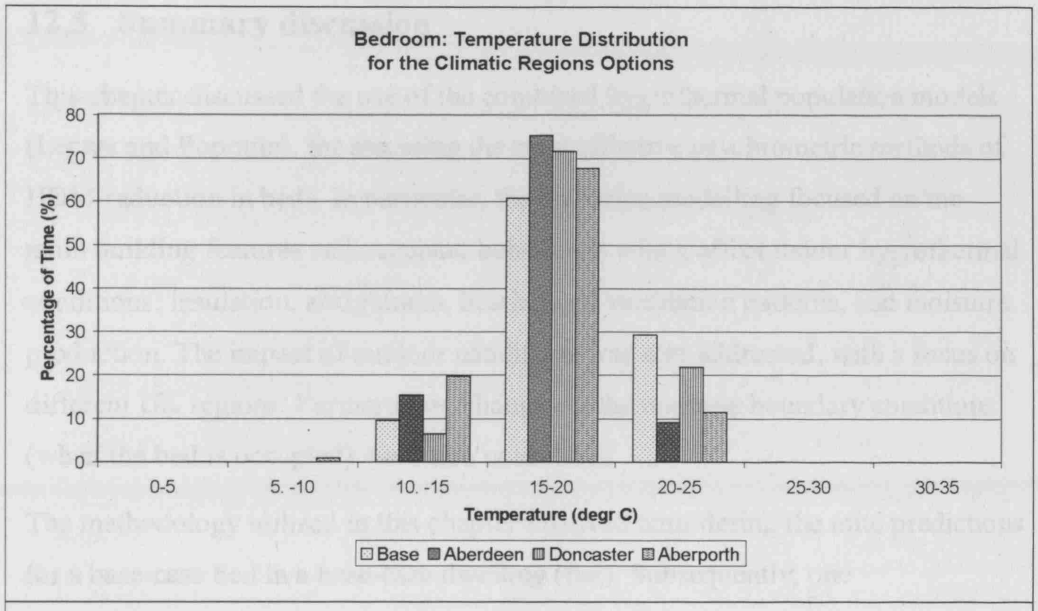
Figure 12.4.5 and 12.4.6 show the frequency distribution of the temperatures and RHs for the 3 outdoor conditions, and for the base-case. The average conditions are provided in Table 12.4.4.

Table 12.4.4 Average hygrothermal conditions over 12 months for the scenarios focusing on outdoor conditions, in each of the selected mattress cells.

	Temp. (°C)			RH (%)		
Scenarios	**Cell A**	**Cell B**	**Cell C***	**Cell A**	**Cell B**	**Cell C***
Base-case	23.6	20.4	18.8	55.7	56.1	57.6
20) Climatic region: Aberdeen	22.7	19.4	17.6	56.8	57.0	58.6
21) Climatic region: Doncaster	23.4	20.3	18.6	56.6	56.8	58.2
22) Climatic region: Aberporth	22.8	19.3	17.4	56.6	57.2	59.5

*Cell C has the same hygrothermal conditions as the bedroom.

[6] Lower temperature and lower vapour pressure excess (worse for mites)
[7] Higher temperature and higher vapour pressure excess (better for mites)



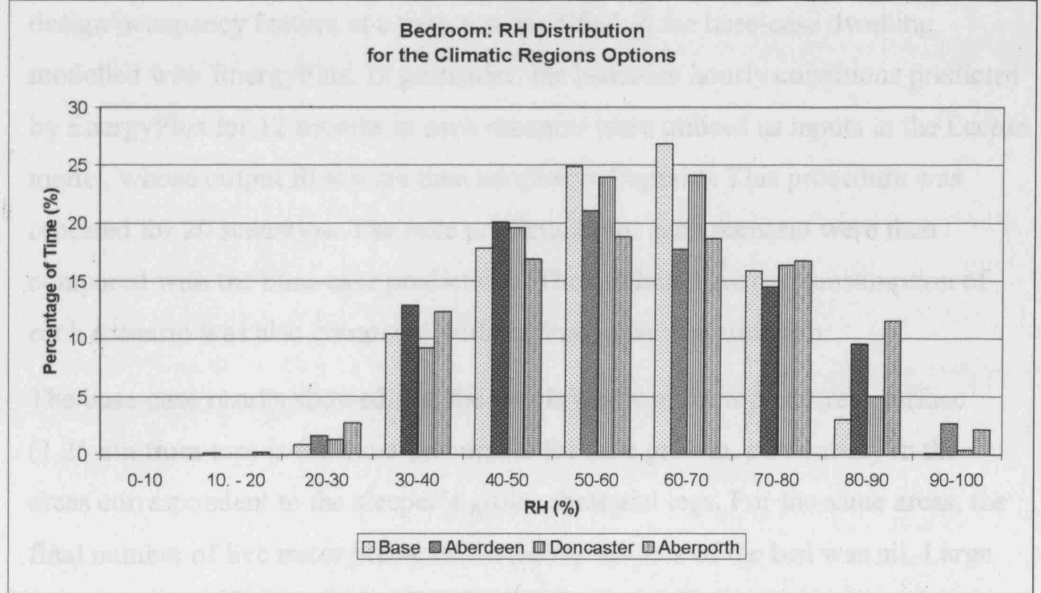


Fig 12.4.5 and 12.4.6 Temperature and RH distribution over 12 months for the three outdoor conditions options and for the base-case.

The graphs show that Aberdeen and Aberporth have higher RHs (>80%), but also lower RHs (<40%) than the base-case. The main relevant difference between Doncaster and the base-case appears to be that the former has lower temperatures (<20 °C) than the latter, with nearly 70% of the Doncaster temperatures falling between 15-20 °C.

The next section is a summary discussion of the scenarios modelling results.

12.5 Summary discussion

This chapter discussed the use of the combined hygrothermal population models (Lectus and Popmite), for assessing the most effective psychrometric methods of HDM reduction in beds. In particular, the scenarios modelling focused on the main building features and occupant behaviours which affect indoor hygrothermal conditions: insulation, airtightness, heating and ventilation patterns, and moisture production. The impact of outdoor conditions was also addressed, with a focus on different UK regions. Furthermore, changes in the mattress boundary conditions (when the bed is occupied) were also considered.

The methodology utilised in this chapter involved considering the mite predictions for a base-case bed in a base-case dwelling (flat). Subsequently, one design/occupancy feature at a time was modified in the base-case dwelling, modelled with EnergyPlus. In particular, the bedroom hourly conditions predicted by EnergyPlus for 12 months in each scenario were utilised as inputs in the Lectus model, whose output files were then adopted in Popmite. This procedure was repeated for 20 scenarios. The mite predictions for each scenario were then compared with the base-case predictions. The predicted energy consumption of each scenario was also compared with the base-case consumption.

The base-case results showed that the first layer from the top mattress surface (1.25 cm from top) is the most favourable for mite growth, particularly in the areas correspondent to the sleeper's groin, chest and legs. For the same areas, the final number of live mites predicted on the *top surface* of the bed was nil. Large variations in mite predictions after 12 months were found for the various mattress cells.

The scenarios modelling showed that changes from base-case bedroom conditions differently affect mite predictions for each mattress cells. For example, in one scenario the mite predictions were 32 times higher than base-case in one of the mattress cells, but they were 2539 times higher than base-case in another mattress cell. Furthermore, in some cases a certain scenario caused a *reduction* in predicted mite numbers for one cell, and an *increase* for another cell. This is because Popmite is sensitive to both temperature and RH, and – due to threshold effects -

its sensitivity to changes in hygrothermal conditions differs, depending on the conditions to which these changes are applied to.

This chapter aimed to identify those scenarios which could have the greatest impact on mite growth/reduction in beds. However, it was difficult to establish the exact *order of magnitude* in mite reduction/increase from base-case resulting from the scenarios considered in this chapter, for the following reasons:

- Changes in bedroom conditions affected the mite predictions for the various mattress cells differently. Since mite movement across the mattress cells is not currently modelled in Popmite, it is difficult to determine whether and by how much the mattress *as a whole* will have increased/decreased mite levels. Currently, there is insufficient information on mite movement, especially under transient conditions. More research is required on how hygrothermal conditions, food and space availability might affect mite movement in a bed, in order to be able to simulate properly the overall effect of changes in bedroom conditions on the mattress.
- Some scenarios considered in this chapter resulted in rather large increases of mite predictions from base-case results. However, in real mattresses the lack of food or space will at some point restrict population growth, and/or lead to mite migration to other areas (Wilkinson et al., 2002). Some (limited) information is available on mite feeding regimes for steady-state conditions. However, more research is required for mite feeding habits under transient conditions. This is particularly crucial for Popmite, which proved most sensitive to input hygrothermal conditions *and* to feeding rates (Chapter 9). More information is also required on how space availability affects mite populations' growth (and movement). Finally, information is also required on typical food densities and distributions found in real mattresses.
- Popmite appeared to over-predict fieldwork results (mites caged in "mite bags"), particularly at high RHs (Chapter 7 and 10). Therefore, the scenarios modelling results have to be taken with some caution (i.e. not in absolute terms), particularly for those scenarios which high RH levels. However, mite predictions should be more reliable for those cases where the RHs (or final predicted mite numbers) are very low.

- The size of the initial population can affect final predictions, as demonstrated by Wilkinson *et al.* (2002), and confirmed in this Chapter. Therefore, more information is required on the distribution and the density of live mites in real beds. However, this information is currently unavailable because of difficulties associated with sampling live mites (Chapter 2).

Despite all the above limitations, the scenarios modelling still gave some indications of those options which might reduce mite numbers, from base-case values:

- Higher fabric permeability;
- Windows open all night in the bedroom;
- Prolonged use of extract fans;
- Increased temperature for the thermostat setting;
- Reduced moisture production rates.

Unfortunately, most of these options result in increased energy consumption. Once the effect of mite movement and of food/space restrictions can adequately be modelled in Popmite/Lectus, it should be possible to select those options which result in the greatest reduction of mite numbers in a bed, with the least energy penalties. It should be highlighted that the options listed above are relevant to the base-case examined in this study, and they may not be as effective for different settings (e.g. greater ventilation rates may be not as effective in a dwelling with large infiltration rates).

Although mechanical ventilation with heat recovery (MVHR) is often advocated as an effective mite eradication method, the results show that some caution might be needed when utilising this system. This is because the ventilation rates achieved by some MVHR systems might not be adequate to sufficiently reduce RH levels, particularly during critical moisture-production times such as cooking, bathing etc. In addition, MVHR also lead to greater temperatures, which might decrease mite development times. Therefore, careful design of the MVHR system might be required, in order to reduce mite numbers.

A range of mattress boundary conditions had been found in the fieldwork study. This range is due to the variability in temperature and moisture production during

sleep, both within and across individuals (Chapter 5). In this chapter it was found that the extremes of these range of boundary conditions can lead to rather dramatically different predictions, for the same bedroom conditions. Therefore, it may be desirable to consider this *range* of conditions, as opposed to the current *fixed* boundary conditions in Lectus.

As expected, given the same building characteristics and occupant behaviours, mite predictions vary in relation to different climatic regions in the UK.

Finally, this chapter (and Chapter 9) showed that changes in hygrothermal conditions can lead to dramatically different Popmite predictions, depending on the hygrothermal conditions to which those changes had been applied. Therefore, it should be emphasised that when Popmite is utilised in scenarios modelling, the choice of appropriate base-case conditions is crucial, in order to obtain valid results.

The results of this Chapter highlighted how the control of house dust mites via hygrothermal means is a complex problem, and this may help to explain why many of the field trials of HDM control (e.g. with MVHR) have yielded conflicting results.

Chapter 12: References

BSI, (2002), *BS 5250: 2002. Code of practice for control of condensation in buildings*, London: BSI.

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

Cunningham MJ, Roos C, Gu L, Spolek G, (2004), Predicting psychrometric conditions in biocontaminant microenvironments with a microclimate heat and moisture transfer model - description and field comparison, *Indoor Air*, 14 (4): 235-242.

Howieson SG, Lawson A, McSharry C, Morris G, McKenzie E, Jackson J, (2003), Domestic ventilation rates, indoor humidity and dust mite allergens - Are our homes causing the asthma pandemic?, *Building Services Engineering Research and Technology Journal*, 24(3): 137-147.

Keresteciogiu A, Swami M, Kamel A, (1990), Theoretical and computational investigation of simultaneous heat and moisture transfer in buildings: "Effective Penetration Depth" theory, *Ashrae Transactions*, 96(1): 447-454.

Meteotest, (1999), *Meteonorm 4.0,* http://www.meteotest.ch/en/mn_home?w=ber, accessed: May 2007.

ODPM (Office of the Deputy Prime Minister), (2006a), *Building regulations, Part L1A: Conservation of fuel and power in new dwellings, Approved Document, 2006 edition*, London: NSB.

ODPM (Office of the Deputy Prime Minister), (2006b), *Building regulations, Part F: Ventilation, Approved Document, 2006 edition*, London: NSB.

Palmer J, Orme M, Pane G, Ridley I, Davies M, Oreszczyn T, Lowe R, (2007), *Investigation of ventilation effectiveness,* (Building Regulations Research, CI 71/6/44, BD2523), Final Report, Version 0.4.

Pretlove SEC, Oreszczyn T, Ridley I, Wilkinson T, Crowther D, (2005), A steady-state model for predicting hygrothermal conditions in beds in relation to house

dust mite requirements, *Building Services Engineering Research and Technology Journal*, 26(4): 301-314.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (submitted), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air.*

Stephen R, (2000), *Airtightness of UK dwellings, BRE Information Paper IP1/00*, Garston (UK): BRE.

Svennberg K, Claesson J, Hansen K, (2005), *Moisture penetration in a chair seat as a response to daily RH variations in the indoor air*, http://www.byv.kth.se/avd/byte/reykjavik/pdf/art_114.pdf, website accessed: May 2007.

Trechsel HR ed, (1994), *Moisture control in buildings*, Philadelphia: The American Society For Testing and Materials.

US Department of Energy, (2007a), *EnergyPlus Version 2*, http://www.eere.energy.gov/buildings/energyplus/, website accessed: May 2007.

US Department of Energy, (2007b), EnergyPlus Testing and Validation, http://www.eere.energy.gov/buildings/energyplus/testing.html, website accessed: May 2007.

US Department of Energy, (2007c), *Weather Files for EnergyPlus*, http://www.eere.energy.gov/buildings/energyplus/cfm/weather_data.cfm, website accessed: May 2007.

Wilkinson T, Horwood J, Cox P, Crowther D, Ridley I, Pretlove S, Oreszczyn T, (2002), Factors affecting the carrying capacity (K) of a mattress for the house dust mite Dermatophagoides Pteronyssinus (Acari: Pyroglyphidae), (Platform Presentation), *International Congress of Acarology, Merida, Mexico, 2002*, http://www.ucl.ac.uk/bartlett-housedustmites/, accessed: May 2007.

CHAPTER 13:

DISCUSSION AND CONCLUSIONS

CHAPTER 13: DISCUSSION AND CONCLUSIONS

This chapter begins with a summary discussion of this thesis's main findings, including a summary of the original aims and objectives (section 13.1). The main conclusions are presented in section 13.2, and section 13.3 summarises the recommendations for future work.

13.1 Summary discussion

Beds are a crucial source of mite allergens, which play a major role in allergic disease, particularly asthma. House dust mites require a specific combination of hygrothermal conditions to thrive. These conditions depend on a number of interacting factors, such as: climate; building characteristics; heating, ventilation and moisture-producing habits; mattress properties; etc. Because of the complexity of the many interacting factors, a modelling approach is required.

This thesis aimed to test the hypothesis that a combined HDM population-hygrothermal model for beds can adequately predict field data and that the model can be a valuable tool for scenario modelling and intervention studies focused on the psychrometric control of house dust mites in UK housing. In particular, this thesis tested two combined hygrothermal population models: a 'simple' steady-state one-dimensional set of models, combining the hygrothermal model BED (Pretlove *et al.*, 2005) with the population model MPI (Crowther *et al.*, 2006). The other set of combined models was a 'complex' transient 3-dimensional model combining the hygrothermal model Lectus (Ridley *et al.*, submitted) with the population model Popmite (Biddulph *et al.*, 2007).

The thesis aimed to:

1. Establish whether the models' predictions are satisfactory, in relation to fieldwork data;
2. Assess the models' capabilities, including the advantages and limitations of the steady state model versus the transient model.

3. Ascertain the scope for using the models in order to establish adequate design and occupant behaviour strategies for the psychrometric control of house dust mites in UK dwellings.

These objectives were successfully accomplished through a combination of fieldwork and scenarios modelling. The fieldwork study involved testing the models against realistic transient conditions. Also, a novel technique was utilised, whereby live wild DP mites were caged and installed in monitored beds, in order to assess the impact of real hygrothermal conditions on mite populations.

The models tested in this thesis are at the forefront of research in their field. The complex hygrothermal population model (Lectus/Popmite) is the first capable of simulating the effect of realistic transient 3-d hygrothermal conditions on a population of 'wild' mites. Furthermore, no other hygrothermal bed model has been tested against *several monitored real beds*. Finally, the 'mite bag' technique is unique and it allows testing the population model against hygrothermal data from real beds.

The results showed a good agreement between field data and the predictions of the hygrothermal bed models (Lectus and BED), when the uncertainties due to input variables and measurements were taken into account. It was found that for both models the uncertainties in input variables resulted in a smaller uncertainty in model predictions, than the uncertainties due to measurement. Therefore, future studies would benefit from sensors with a greater accuracy than those adopted in this thesis, if one wishes to establish the accuracy of the models further. The sensor should be calibrated under transient conditions, for example to take into account potential differences in response times. This was not possible in this study due to financial constraints. However, the sensors would have to be both compact (to avoid discomfort to participants) and inexpensive, to allow for monitoring a sufficient number of beds.

The comparison between the Lectus predictions and field data showed that *on average* the boundary conditions assumed in Lectus are sufficiently representative of fieldwork data. However, a *range* of boundary conditions occurs in reality, since there is a degree of variability in hygrothermal conditions on the bed surface, both within individuals and across individuals. This variability is due to a

combination of different factors, including: differences in heat and moisture output during sleep within and across individuals; clothing levels; different hygrothermal properties of mattresses, duvets and pillows; movement levels during sleep. Since the boundary conditions utilised in Lectus partly depend on the mattress properties, they should be used with caution when attempting to replicate the exact conditions in a specific bed whose properties are significantly different from those used for the fieldwork study.

At present, the BED model only predicts one location in the mattress, directly under the occupant chest. This was revealed as the least favourable location for mite growth. The scenarios modelling in this thesis suggests that the most favourable location is at 1-2 cm below the top mattress surface, under the groin area, followed by the chest area. It is therefore proposed that the BED model be modified to predict the most favourable environment to mites by incorporating an additional algorithm from the Glazer method (BSI, 2002). The results showed that the predictions calculated in this manner are not significantly different from the corresponding average Lectus predictions. Therefore, potentially the BED model could be utilised in order to calculate monthly hygrothermal conditions for two locations in the mattress, corresponding to the least favourable and one of the most favourable locations[1] for mite growth in beds. However, these predictions should be taken with some caution, since scenarios modelling revealed that space, food and mite movement may be crucial factors for the determination of the number and the distribution of mites in beds. Furthermore, the impact of transient conditions is important, as demonstrated in Chapter 10 and by published information.

The comparison of Popmite predictions[2] with the results from the caged mites (mite bags) resulted in a correlation between measured and predicted results which can be considered adequate (R-square value=0.58) - especially when taking into account the natural variability of biological phenomena. The results also indicated that Popmite is better at predicting juveniles than adults, with a tendency to over-predict juveniles numbers, particularly at high RHs. This suggests that in Popmite mites may be breeding too fast once the hygrothermal conditions are

[1] In the scenarios modelling with Lectus, the most favourable location was the groin area, followed by the chest. The BED model simulates conditions under the chest area.
[2] Popmite version 7d was tested in this thesis

ideal, in comparison with the caged mite results. However, several sources of uncertainty exist in the measurements (e.g. loggers accuracy, mite counting and identification at the beginning and at the end of the monitoring period), as well as in some of the model input variables. A source of uncertainty might also be the age structure of the starting population in the mite bags. The impact of some uncertainties is rather difficult to estimate: e.g. the accuracy with which the acarologist can count/identify the mites, or the age of the mites utilised in the mite bags.

The MPI predictions fitted the field measurements less accurately than Popmite predictions (MPI R-squared value: 0.49). This was somewhat expected, since the mite bags were kept under transient conditions, whilst MPI is a steady-state model. Furthermore, the experiments which formed the basis of the MPI model included a larger population of laboratory-reared mites, as opposed to a smaller population of *wild* mites on a "natural" diet, as in the fieldwork study. The MPI model - like Popmite - tends to over-predict measurements by a factor of 1.5. These over-predictions mostly occur at high RHs, whilst for mid-range values the MPI model has a tendency to under-predictions.

The sensitivity analysis showed that all 4 models tested in this thesis are sensitive to input hygrothermal conditions. The sensitivity of the population models to changes in hygrothermal conditions also depends on the hygrothermal conditions to which these changes are applied. Temperature – not only RH – plays an important role on the population models, particularly Popmite. Strong threshold effects could be observed for both population models. The largest variations in Popmite predictions are not only produced by changes in hygrothermal conditions, but also by changes in the eating rate. Since little published information is available on this parameter, further research on this issue is essential.

Some striking similarities occur between Popmite and MPI predictions. For example, both Popmite and MPI over-predict mite bags measurements, particularly at high average RHs. Also, at high RHs there are some noticeable variations in the differences between predictions and measurements at similar RHs, for both models. These similarities are reassuring, since the two models were developed independently and with radically different approaches. The MPI

is a simple empirical model, while Popmite is a complex model, using very different empirical data. At present it is unclear what might be causing these over-predictions. These discrepancies between predictions and measurements might occur because mite growth is potentially more difficult to predict than death/survival, since the latter is an on/off event, while even a small error in growth rates might be amplified over time - leading for example to over-predictions. However, this is unlikely, since the similarities between the models' predictions at high RHs are a little too striking. One possibility is that the caged mites do not thrive in the bags as well as they would in a "natural" environment. Mite counting might also be more difficult with high mite numbers, which could lead to under-estimation and/or inaccurate results. In order to determine whether this might be the case, further experiments should be carried out on the mite bags. However, another explanation for the population models' over-predictions might be that the models do not explicitly include restrictions for food or space availability, which could occur in mite bags. Competition with moulds might also occur at high RHs, which could affect mite growth in the mite bags but it is not explicitly simulated in the models.

This thesis also described a pilot longitudinal intervention study on the effectiveness of house dust mite allergen avoidance for twelve asthmatic children (two being controls). In addition to mite allergen removal, the study included tailored advice aimed at reducing future mite population growth via changes in moisture production, heating and ventilation habits. The post-intervention results showed that there was a statistically significant ($p<0.01$) decrease of average measured RHs in the bedrooms, even when taking into account the effect of changes in outdoor conditions (average difference between pre and post adjusted average bedroom RH: 5.1%). The population modeling results indicated that during the pre-intervention period the mite populations were rather stable (average MPI $\cong$ 1), suggesting that even small hygrothermal changes could determine whether the population grows or declines. The study showed that it can be difficult to control/assess changes in hygrothermal behaviours. In order to facilitate this, ventilation rates and air infiltration should both be measured. In future studies the frequency of window opening and of extractor fans usage should ideally also be measured, both in the pre and the post intervention periods.

Also, the selection of similar building types for the study would make the results more comparable, facilitating the assessment of the interventions' effectiveness. The study also demonstrated the importance of hygrothermal and HDM population models in helping to interpret the results of such intervention studies. The models might also help to explain some of the contradictory results often obtained in field trials of HDM psychrometric control measures.

This thesis also discussed the use of the combined hygrothermal population models (Lectus and Popmite), for assessing the most effective psychrometric methods of HDM reduction in beds. In particular, the scenarios modelling focused on the main building features and occupant behaviours which affect indoor hygrothermal conditions: insulation, airtightness, heating and ventilation patterns, and moisture production. The impact of outdoor conditions was also addressed, with a focus on different UK regions. Furthermore, changes in the mattress boundary conditions (when the bed is occupied) were also considered. The results showed that the first layer from the top mattress surface (1.25 cm from top) is the most favourable for mite growth, particularly in the areas correspondent to the sleeper's groin, chest and legs. For the same areas, the final number of live mites predicted on the *top surface* of the bed was nil. Large variations in mite predictions after 12 months were found for the various mattress cells.

The scenarios modelling showed that changes from base-case bedroom conditions differently affect mite predictions for each mattress cells. For example, in one scenario the mite predictions were 32 times higher than base-case in one of the mattress cells, but they were 2539 times higher than base-case in another mattress cell. Furthermore, in some cases a certain scenario caused a *reduction* in predicted mite numbers for one cell, and an *increase* for another cell. This is because Popmite is sensitive to both temperature and RH, with strong threshold effects.

The scenarios modelling aimed to identify those scenarios which could have the greatest impact on mite growth/reduction in beds. However, it was difficult to establish the exact *order of magnitude* in mite reduction/increase from base-case. Since predicted mite numbers varied so widely across the mattress for the same bedroom conditions, it would be necessary to assess whether and by how much the mattress *as a whole* will have increased/decreased mite levels, from the base-case. However, due to lack of published information, Popmite does not include

mite movement across the cells. This movement might be driven by lack of food/space, which will also limit the population growth. In other words, the carrying capacity of a mattress is not only affected by the hygrothermal conditions to which the mattress is exposed, but also by food and space availability, as well as by mite movement (Wilkinson *et al.*, 2002). These additional parameters, however, are not currently modelled in Popmite, due to lack of detailed information. Therefore, some of the scenarios modelling predictions might overestimate the effect of favourable hygrothermal conditions on mite populations in a bed. This risk of over-predictions is reinforced by the fact that Popmite appears to over-predict field results, particularly at high RHs. Therefore, the scenarios modelling results which are most likely to be true are those where the final population is zero (i.e. all mites have died), due to unfavourable hygrothermal conditions.

Despite all the above limitations, the scenarios modelling still gave some indications of those options which *reduced* mite numbers, from base-case values: higher fabric permeability; windows open all night in the bedroom; prolonged use of extract fans; increased temperature for the thermostat setting; reduced moisture production rates. However, it should be emphasised that these options might lead to different mite predictions, if applied to a significantly different base-case dwelling (e.g. greater ventilation might not be as effective in a base-case dwelling with large infiltration rates). Although mechanical ventilation with heat recovery (MVHR) is often advocated as an effective mite eradication method, the scenarios modelling results show that some caution might be needed when utilising this system.

The following section lists the main conclusions of this thesis.

13.2 Conclusions

Based on the findings described in this thesis, the following conclusions can be drawn:

1. The predictions of the hygrothermal bed models (BED and Lectus) show good agreement with field data, when the uncertainties due to input variables and

measurements are taken into account. However, areas for model improvement have also been identified.

2. The bed simulated in Lectus is representative of average conditions with a "typical" sprung mattress. It should be highlighted that Lectus cannot - and has not been designed to - simulate specific mattresses with complicated designs, nor it is fully representative of the variability of hygrothermal conditions occurring in real beds. Lectus is however very useful to assess the *average* effect that room conditions might have on an *average* occupied bed.
3. The fieldwork study gave an indication of the likely *range* of boundary conditions occurring in a sprung mattress during occupation, due to a variability both within and across individuals. Boundary conditions are one of those input variables to which Lectus is sensitive, and the scenarios modelling revealed that the extremes of these range in boundary conditions can lead to rather different mite predictions, for the same bedroom conditions. Therefore, it may be desirable to utilise this *range* of boundary conditions (i.e. worst and best case scenarios) when using Lectus for scenarios modelling.
4. The results for the population models showed that Popmite predictions have a better agreement with fieldwork results (R-squared value: 0.58) than MPI's predictions (R-squared value: 0.49). Therefore, if transient conditions are available, Popmite should be used, particularly if the average RH is close to the Critical Equilibrium Humidity. Hence, as laboratory studies show, less accurate results are obtained when using steady-state average conditions as opposed to transient conditions. However, if only constant conditions are available, MPI is marginally better than Popmite (R-squared value: 0.47).
5. Both the mattress and the population models tested in this thesis are sensitive to input hygrothermal conditions. The sensitivity of the population models to changes in hygrothermal conditions depends on the hygrothermal conditions to which such changes are applied (threshold effects). In particular:
    5.1. The mattress models are mostly sensitive to room conditions, followed by boundary conditions for the top mattress surface when the bed is occupied, and to the length of time the bed is occupied (to a lesser extent).

5.2. In most cases, the MPI model is more sensitive to changes in RH, than to changes in temperature. However, depending on the base-case hygrothermal conditions and on the size of the change, changes in temperature can be important as well - particularly at low base-case RHs. The Popmite model is at least as sensitive to changes in temperature as to changes in RH, and in some cases changes in temperature have the largest impact.

5.3. In both population models threshold effects can be observed, often in unpredictable ways. For example, a 10% increase in RH does not lead to a fixed increase in predictions, since this figure depends on the initial hygrothermal conditions to which this 10% increase in RH is applied. Furthermore, an increase in temperature might lead to a decrease or increase in predictions, depending on the hygrothermal conditions to which such changes are applied. Because of these threshold effects, it is not possible to identify a typical change in predictions due to certain hygrothermal changes. The threshold effects may also partly explain why some field trials of the psychrometric control of house dust mites have been inconclusive.

5.4. The largest variations in Popmite predictions are not only produced by changes in hygrothermal conditions, but also by changes in the mites' eating rates. The sensitivity of Popmite also changes in relation to different output types (i.e. adults, juveniles, eggs).

6. Whenever possible, it is advisable to utilise the set of complex models (Lectus-Popmite), as opposed to the set of simple models (BED-MPI). This is for two main reasons: 1) Popmite predicts the effects of transient hygrothermal conditions on mite populations better than the MPI model (which is in fact a steady-state model). Conditions in dwellings are transient, and therefore Popmite should produce more realistic predictions. 2) The BED model only predicts a few locations within the mattress, whilst the scenarios modelling with Lectus showed that the number of predicted mites can vary dramatically, depending on mattress locations.

7. Both Popmite and MPI over-predict the fieldwork measurements (mite bags), particularly at high average RHs. Also, at high RHs there are some noticeable variations in the differences between predictions and measurements at similar RHs, for both models. Further investigation is required to assess whether these similarities are associated, for example, with anomalies in the mite bag results (see the following section on future work). However, another explanation for the population models' over-predictions might be that the models do not explicitly include restrictions for food or space availability, nor competition with moulds, which occurred in the mite bags.

8. The models can be rather useful in intervention studies, as demonstrated by the pilot described in this thesis. The models can be utilised, for example, to identify those dwellings most at risk of mite infestation and therefore help prioritise the interventions or allocate the case/control status. Furthermore, the population model Popmite might be utilised to assess the impact of psychrometric interventions on mite populations, since the latter might have been eradicated by allergen removal strategies. Because of threshold effects in Popmite, if *adjusted* hygrothermal conditions are utilised in Popmite in order to exclude the effect of changes in outdoor conditions, then the results should be considered bearing in mind the strong threshold effects characterising Popmite.

9. The first layer from the top mattress surface (1.25 cm from top) is the most favourable for mite growth, particularly in the areas correspondent to the sleeper's groin, chest and legs. For the same areas, the final number of live mites predicted on the *top surface* of the bed are nil. Large variations in mite predictions after 12 months can be found for the various mattress cells.

10. The ultimate goal of the models tested in this thesis is the identification of those building design and occupant behaviour features which could have the greatest impact on mite growth/reduction in beds. The scenarios modelling revealed that changes in bedroom conditions affect the number of predicted mites for the various mattress cells differently. Therefore, it is necessary to assess the mattress's overall mite carrying capacity, in order to determine whether changes in bedroom conditions will result in an overall reduction or increase in a bed's mite numbers. However, due to lack of published

information, Popmite currently simulates only the effect of one of the factors affecting a mattress carrying capacity (i.e. hygrothermal conditions), whilst the other factors are: food and space availability, as well as mite movement. This lack of information represents a potentially significant source of uncertainty in the scenarios modelling predictions. An additional source of uncertainty is represented by the tendency of Popmite to over-predict, particularly at high RHs. Due to these uncertainties, the scenarios modelling predictions should not be taken at face value, but cautiously and in relative terms – especially for favourable hygrothermal conditions. The scenarios resulting in *nil* mite predictions should be more reliable than the scenarios where mite numbers increase significantly.

11. Despite the limitations outlined in the previous point, the scenarios modelling gave some indication of those options which can reduce mite numbers in a bed located in a dwelling compliant with current Building Regulations: higher fabric permeability; windows open all night in the bedroom; prolonged use of extract fans; increased temperature for the thermostat setting; reduced moisture production rates. Unfortunately, most of these options result in increased energy consumption. Once the effect of mite movement and of food/space restrictions can adequately be modelled, it should be possible to select those options which result in the greatest reduction of mite numbers in a bed, with the least energy penalties.

12. Although mechanical ventilation with heat recovery (MVHR) is often advocated as an effective mite eradication method, the results show that some caution might be needed when utilising this system. This is because the ventilation rates achieved by some MVHR systems might not be adequate to sufficiently reduce RH levels, particularly during critical moisture-production times such as cooking, bathing etc. In addition, MVHR also leads to greater temperatures, which might accelerate mite development. Therefore, careful design of the MVHR system might be required, in order to reduce mite numbers. This may in part explain why field trials with MVHR have been inconclusive.

13. Popmite users for scenarios modelling purposes should be aware that if the hygrothermal input data start with late summer/early autumn months,

predictions of mite numbers will be higher than the case where the hygrothermal input data start with winter months. Furthermore, the initial population size might determine whether the final population is eradicated.

14. Changes in hygrothermal conditions can lead to dramatically different Popmite predictions, depending on the hygrothermal conditions to which those changes had been applied. Therefore, it should be emphasised that when Popmite is utilised in scenarios modelling, the choice of appropriate base-case conditions is crucial, in order to obtain valid results.

13.3 Recommendations for future work

This section lists the recommendations for future work, based on this thesis findings:

1. In order to identify with more certainty those building design and usage features which greatly affect mite growth/decline, it is necessary to determine a mattress overall mite carrying capacity. For doing so, further studies are recommended on the impact of food and space availability on the size of a population of wild DP mites, including the impact of hygrothermal conditions on food consumption. This issue is particularly important for Popmite, due to its sensitivity to eating rates. Furthermore, the eating rates are linked to faeces production (i.e. allergen), which has a direct impact on respiratory health.
2. Both population models tested in this thesis showed a tendency to over-predict the fieldwork results (mite bags), particularly at high RHs. Also, at high RHs there were some noticeable variations in the differences between predictions and measurements, for both models. At present it is unclear what might be causing these over-predictions. One possibility is that the caged mites do not thrive in the bags as well as they would in a "natural" environment. Mite counting might also be more difficult with high mite numbers, which could lead to under-estimation and/or inaccurate results. In order to determine whether this might be the case, further experiments should be carried out, where two mite populations with the same size are held in identical hygrothermal conditions, but one population is kept in mite bag(s), and another population is held in a more "natural" environment. Careful thought

should be given on the amount of food provided to each population. The possible impact of the competition with moulds at high RHs should be taken into account. A calibration protocol for mite counting should also be developed and adopted in all future studies utilising the mite bags.

3. The age structure of the population in the mite bags might be a confounding factor when comparing predictions with mite bags results. Future studies should investigate the impact of different numbers and population structures in the mite bags results. Ideally, the number of mites in the bags should be increased, in order to make the mite bag sample more representative of the population (e.g. spread of all ages).

4. At present Popmite predictions are linked to egg-laying rates. However, eggs are difficult to count and therefore Popmite predictions for eggs could not be tested against field data. Further research is required on egg-laying rates under different hygrothermal conditions and for different food availabilities, as well as on new methods for identifying/counting mite eggs.

5. The Lectus model was developed under laboratory conditions and was tested in this thesis mostly during autumn and winter months. It may be desirable to test the model during summer times, where different room conditions might affect the boundary conditions, especially in the case of high outdoor temperatures.

6. When testing the Lectus and the BED model, the uncertainties in the fieldwork measurement were greater than those in input variables. If it is deemed useful to asses the models' validity with greater accuracy - for example once the population model(s) are developed further – sensors with greater accuracy than those utilised in this thesis are required. Since hygrothermal conditions in a bed can change quite quickly, it is recommended that any logger utilised in the mattress is calibrated under transient conditions, for example to account for potential differences in response times between sensors. Also, if greater accuracy is required for Lectus, the use of a greater number of mattress surface sensors is recommended, as well as recording the exact bed occupancy times. Standardisation of duvet and pillows may also contribute in reducing the scope for differences across participants. However,

it would also be important to determine to what extent boundary conditions change, if different duvet/pillows/mattresses are utilised. In order to do so, each participant should be monitored several times, utilising different beddings and mattresses under similar room conditions.

7. The BED model currently predicts the hygrothermal conditions of a mattress area least favourable to mite growth. It is recommended that the BED model is modified so that it can also predict hygrothermal conditions for the mattress zone which is more favourable to mite growth (1-2 cm below the top mattress surface). Once the issues associated with food/space availability and mite movement are clarified, Lectus and Popmite should be utilised to examine further the mite spatial distribution and the carrying capacity of beds. This information could then be adopted to modify the BED/MPI models, in order to make their predictions more realistic.

8. The development of the combined hygrothermal population models was originally driven by the impact of dust mite allergens on respiratory health. However, currently a knowledge gap still exists on the exact mechanisms associated with asthma development and exacerbation, including the impact of HDM allergen exposure on HDM sensitisation, and the impact of HDM sensitization on asthma development/exacerbation. Some studies have generated some doubts on the concept of a linear dose-response relationship between HDM allergen exposure and HDM sensitisation, suggesting the possibility of a *bell-shaped* relationship. If that was the case, then a reduction in HDM allergen levels could result in an *increase* in sensitisation rates, depending on the initial allergen levels. This is a crucial question for any research on HDM allergen avoidance - including the psychrometric control of house dust mites – and it needs to be further investigated.

9. Most epidemiological studies on HDM allergen exposure, sensitisation, asthma development or asthma exacerbation have reported their findings in terms of HDM allergen levels. Therefore, it is desirable that the population models tested in this thesis are developed further, in order to predict allergen levels, as opposed to populations only. This would be particularly crucial if it was found that the relationship between HDM allergen exposure and sensitisation is bell-shaped, since this would require an assessment of the

exact levels of allergen production, as opposed to an assessment of growth/decline in such levels. The impact of cleaning regimes and the role of the reservoir effect might also have to be investigated.

Chapter 13: References

Biddulph P, Crowther D, Leung B, Wilkinson T, Hart B, Oreszczyn T, Pretlove S, Ridley I, Ucci M, (2007), Predicting the population dynamics of the house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle, *Experimental and Applied Acarology*, 41(1-2): 61-86.

BSI, (2002), *BS EN ISO 13788:2002. Hygrothermal performance of building components and building elements. Internal surface temperature to avoid critical surface humidity and interstitial condensation. Calculation methods.* London: BSI.

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I, (2006), A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae), *Experimental and Applied Acarology*, 39: 127-148.

Pretlove SEC, Oreszczyn T, Ridley I, Wilkinson T, Crowther D, (2005), A steady-state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements, *Building Services Engineering Research and Technology Journal*, 26(4): 301-314.

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M, Hart B, (submitted), A model to predict the hygrothermal conditions experienced by House Dust Mites within beds, submitted to: *Indoor Air.*

Wilkinson T, Horwood J, Cox P, Crowther D, Ridley I, Pretlove S, Oreszczyn T, (2002), Factors affecting the carrying capacity (K) of a mattress for the house dust mite Dermatophagoides Pteronyssinus (Acari: Pyroglyphidae), (Platform Presentation), *International Congress of Acarology*, Merida, Mexico, 2002, http://www.ucl.ac.uk/bartlett-housedustmites/, accessed: May 2007

Volume 2: Appendix

The Psychrometric Control of House Dust Mites: Testing the Validity in UK Dwellings of Two Combined Hygrothermal Population Models for Beds

Marcella Ucci

The Bartlett School of Graduate Studies

University College London

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University College London

University of London

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Appendix to Chapter 2

A.2: House dust mites, atopy and asthma

Atopy is strongly associated with diseases such as asthma, hay fever, eczema and rhinitis, but not all atopic individuals develops clinical manifestations of allergy, nor everyone with a clinical syndrome compatible with allergic disease can be proven atopic when tested for specific IgE for common environmental allergens. This is particularly true for asthma (Jarvis and Burney, 1998). Asthma is the most serious of allergic diseases, being disabling and occasionally fatal. Many studies – especially cross-sectional - have demonstrated that asthma is strongly associated with atopy, particularly in children (Cole Johnson *et al.*, 2002). Therefore, a theoretical paradigm has often been advocated in which allergen exposure produces atopic sensitisation in susceptible individuals, and continued exposure then leads to clinical asthma through the development of airways inflammation, bronchial hyperresponsiveness and reversible airflow obstruction (Pearce *et al.*, 1999). However, not all asthma cases can be *attributed* to atopy, since the association between atopy and asthma may not reflect causality, at least in some cases. For example, inherited genetic factors could increase susceptibility both to asthma and to the production of raised IgE levels; higher total IgE levels could in part be a consequence of asthma itself (Pearce *et al.*, 1999). In this section the relationship between asthma and atopy is discussed further, with a particular focus on house dust mites.

Some authors have questioned the extent to which the development of asthma is attributable to atopy. Pearce *et al.* (1999) calculated the population attributable fraction for atopy and asthma, based on the review of several studies. The population Attributable Fraction (AF) can be defined as the proportion of disease cases over a specified time that would be prevented following the elimination of the exposures, assuming the exposures are causal (Rockhill *et al.*, 1998). If exposure (atopy in this case) has an odds ratio for asthma of R, the proportion of exposed cases (i.e. atopic asthmatics) that are attributable to exposure (i.e. atopy) is:

$AF_{exp}=(R-1)/R$

The proportion of all cases (i.e. asthmatics) in the general population that are attributable to exposure (atopy) is the population attributable fraction, which is:

$AF_{pop}=P*(R-1)/R$

where P is the proportion of all cases that are exposed (i.e. proportion of atopic asthmatics).

In their review Pearce *et al.* (1999) found that the proportion of cases attributable to atopy partly depends on the definition of atopy itself. If atopy is defined in a more stringent way (for example, four or more positive skin prick tests, rather than just one), the association with asthma *increases* (as reflected in the relative risk estimate), but since the proportion of atopic asthmatics decreases, the population attributable risk *decreases*. Pearce *et al.* concluded that the population-based proportion of asthma cases that attributable to atopy is usually less than 50%, varying from one third to two thirds, depending on the definition of atopy. However, Pearce *et al.* also emphasised that the studies may have differed methodologically, and they were not all carried out in the same population.

Sunyer *et al.* (2004) reported geographic variations in the effect of atopy on asthma in the European Community Respiratory Health Study (ECRHS). The ECRHS was the first international multicenter study in adults (20-44 years old) using a common standard protocol measuring atopy and asthma in the same time period (1990-1994). In the study, atopy was defined as the presence of IgE sensitisation to any allergen. The results from the study are summarised in Table A.2.1.

TABLE A.2.1 (Sunyer *et al.*, 2004) ECRHS (adults, aged 20-44): Prevalence of asthma and specific IgE by center (range in countries with 2 or more centers) and association (odds ratio or range of odds ratios in countries) between asthma and specific IgE at the individual level by center (number of individuals = 13,558; number of centers = 36)

Countries ordered by % of atopy* (no. of centers)	Prevalence, % (95% CI)					Odds ratio (95% CI)↔			
	Asthma	House dust mite	Cat	Timothy Grass	Atopy*	House dust mite	Cat	Timothy Grass	Atopy*
Estonia (1)	7	9	5	9	18	1.82	8.74	3.12	1.25
Iceland (1)	3	9	7	12	23	8.91	7.02	4.59	4.21
Spain (5)	4-11	7-28	3-13	9-20	17-42	1.48-4.54	2.78-8.90	1.62-4.02	1.33-5.44
Norway (1)	7	14	7	15	26	3.17	5.46	2.76	5.16
Italy (3)	6-15	11-13	4-7	12-21	24-30	2.53-5.30	1.10-9.51	2.76-5.42	2.94-4.85
Sweden (3)	8-10	7-12	13-14	17-18	30-32	1.88-2.36	2.60-5.54	2.02-3.58	1.92-5.17
France (4)	6-13	18-35	7-10	12-20	29-43	1.79-4.64	3.43-6.48	1.37-3.98	1.53-4.60
Belgium (2)	5-9	22-27	9-9	16-17	35-36	3.65-3.65	2.78-5.03	4.17-5.10	4.24-5.28
Germany (2)	3-7	16-19	8-11	21-25	35-40	0.23-2.55	2.60-4.47	1.35-2.55	1.36-3.31
United Kingdom (4)	9-14	20-28	8-14	13-27	34-44	2.01-5.07	2.33-5.17	1.62-2.86	2.04-3.93
The Netherlands (3)	5-7	24-29	6-10	17-22	36-41	2.06-6.14	3.75-5.52	2.44-5.49	2.03-5.74
Ireland (1)	12	35	7	17	41	3.15	3.62	5.51	2.07
New Zealand (3)	11-14	31-33	6-13	23-33	40-46	1.74-6.14	0.83-8.34	2.19-3.14	1.57-4.58
United States (1)	12	19	13	34	43	1.01	2.13	2.48	2.52
Switzerland (1)	10	19	15	33	45	1.86	1.31	1.75	1.53
Australia (1)	12	32	9	29	45	2.89	3.24	2.41	3.22
All (95% CI),	9 (8-10),	21 (18-23),	8 (7-10)	19 (17-21),	34 (31-37),	2.78	4.18	2.63	2.82
#p value for heterogeneity	<.001	<.001	<.001	<.001	<.001	(2.41-3.20), .14	(3.54-4.93), .45	(2.30-3.02), .92	(2.44-3.28), .15

*Any: house dust mite, cat, timothy grass, C herbarum, and birch, P judaica, or ragweed. #Estimated with meta-analysis. ↔Adjusting for age, sex, and smoking.

The results show that the prevalence of specific IgE sensitisation varied widely between centres, even within the same country (e.g. dust mites in Spain or in France). The association between IgE sensitisation and asthma was strong, but the associations were not statistically significantly heterogeneous across centres, for any allergen. Sunyer *et al.* also calculated the proportion of asthma cases attributable to atopy and to specific allergens in the various centres. The results are summarised in Table A.2.2.

Table A.2.2 (Sunyer *et al.*, 2004). ECRHS (adults, aged 20-44): AF of asthma, defined on the basis of symptoms, caused by specific IgE sensitization and atopy by center

Countries ordered by % of atopy*	Center	HDM	Cat	Tim. grass	Atopy* (95% CI)
Estonia	Tartu	6	17	13	4 (219.0 to 22.0)
Iceland	Reykjavik	35	28	25	40 (22.1 to 64.5)
Spain	Albacete	3	9	7	11 (24.8 to 24.8)
	Oviedo	10	6	15	25 (25.9 to 46.6)
	Galdakao	40	23	13	45 (0.0 to 70.2)
	Huelva	14	22	10	9 (227.3 to 35.5)
	Barcelona (bcn)	32	37	8	61 (227.8 to 88.1)
Norway	Bergen	19	19	18	47 (26.1 to 61.3)
Italy	Pavia	24	0	24	26 (26.1 to 47.8)
	Turin	10	17	20	37 (10.7 to 55.2)
	Verona	21	21	21	44 (6.7 to 66.8)
Sweden	Umea	6	31	26	50 (25.3 to 66.5)
	Goteborg	8	15	13	28 (5.2 to 44.7)
	Uppsala	7	16	20	20 (25.9 to 39.2)
France	Grenoble	12	15	13	16 (216.0 to 39.7)
	Paris	16	18	21	36 (14.4 to 51.6)
	Montpellier	15	11	5	12 (28.8 to 28.3)
	Bordeaux	48	25	23	55 (33.1 to 69.4)
Belgium	South-Antwerp	31	11	27	46 (0.7 to 70.9)
	Antwerp city	37	22	31	55 (17.9 to 75.4)
Germany	Erfurt	214	10	7	11 (226.4 to 37.4)
	Hamburg	19	22	24	43 (19.5, 60.0)
United Kingdom	Cardiff	19	11	11	22 (21.6 to 40.2)
	Ipswich	36	22	23	44 (18.1 to 61.3)
	Norwich	19	20	17	26 (22.2 to 45.9)
	Cambridge	29	12	12	38 (213.8 to 66.6)
The Netherlands	Groningen	54	20	23	58 (13.5 to 79.7)
	Bergen op Zoom	20	15	20	36 (2.1 to 57.7)
	Gellen	19	14	39	26 (217.3 to 53.8)
Ireland	Dublin	35	12	31	26 (29.7 to 50.2)
New Zealand	Hawkes-Bay	14	21	23	14 (229.7 to 43.0)
	Wellington	51	17	18	52 (23.7 to 70.2)
	Christchurch	51	29	30	49 (16.3 to 68.7)
United States	Portland	0	10	29	35 (3.8 to 56.2)
Switzerland	Basel	12	4	17	17 (210.2 to 37.2)
Australia	Melbourne	32	13	25	45 (21.2 to 61.2)
ALL#		18.2 (13.7, 22.4)	14.1 (11.8, 16.3)	17.1 (14.0, 20.1)	30.4 (24.9 to 35.5)
p value for heterogeneity		<.001	.30	.91	.012

*Atopy: IgE sensitization to any of house dust mite, cat, timothy grass, C herbarum, and birch, P judaica, or ragweed.
#AF in the 36 centers estimated with meta-analysis.

The results in Table A.2.2 show that the population attributable fraction of asthma caused by atopy is approximately 30%. The AF_{pop} for atopy could have been underestimated because of the definition of asthma used. The overall AF_{pop} for atopy increased to 42.6% when the diagnosis of asthma was based on wheezing and bronchial hyperresponsiveness, to 45.3% with physician-diagnosed asthma, and 47.9% when patients reported more than 12 attacks of asthma within the past year. The overall AF_{pop} for sensitisation to dust mites was 18.2%. The AF_{pop} for dust mites varied significantly between centres (p value for heterogeneity < 0.001). Sunyer *et al.* concluded that if total elimination from a given population of sensitisation to the allergen considered in their study could be obtained - which is unrealistic – this might possibly result in a reduction of 30% of prevalent asthma cases. Although 56% of asthmatics are sensitised to common aeroallergens, the AF indicates that around 60% of them (i.e. AF among the exposed) would have been prevented by preventing any sensitisation to occur. Sunyer *et al.* highlighted that only geographic variations in the AF for house dust mites were statistically heterogeneous, suggesting that the prevalence of sensitisation to dust mites is the main determinant of geographic variations in the population fraction of asthma attributable to atopy. Sunyer *et al.* conclude that "*IgE sensitisation to common allergens has an effect on asthma prevalence, which varies widely amongst centres. Reasons for the wide variation remains unknown, but they do not seem to be due to the strength of the association between atopy and asthma. Most important appear to be other exposures influencing the expression of asthma among atopic and non-atopic individuals. Levels of allergens in the environment as reflected by the prevalence of atopy – particularly dust mites – also seem to play a role. The present results reinforce the idea that atopy is only one factor in the constellation of factors that play a role in asthma prevalence*". Sunyer *et al.* also point out that interpretation of their results in terms of causality must be taken with caution, since in their study the AF refers to asthma *prevalence* (proportion of *existing* cases, as opposed to *incidence*, i.e. *new* cases). Therefore, their findings are unable to determine whether sensitisation to allergens plays a role in asthma *development*, but only in asthma *prevalence*.

Jaakkola *et al.* (2006) examined the relationship between sensitisation to mites and moulds, and asthma *development* in adults. The study was a population-based incident case-control study, carried out in Finland on adults (21-63 years old) with a total of

485 cases (clinical asthma diagnosis) and 665 controls. The authors found that specific IgE antibodies to *Dermatophagoides Pteronyssinus* and *Acarus Sirus* were not very common, but when detected they were related to significantly increased risk of incident asthma (Odds Ratio for *Dermatophagoides Pteronyssinus*: 2.3; 95% CI: 1.51-3.49). The risk of new asthma increased with increasing IgE antibody levels to *Dermatophagoides Pteronyssinus* (and *Aspergillus fumigatus*). Jaakkola *et al.* also found that the fraction of new adult-onset asthma attributable to IgE antibodies for common aeroallergens was 66% (95% CI, 55-74) among atopic cases. The attributable fraction in the whole working-age population was 30% (95% CI, 23-36). These findings are not dissimilar to those found by Sunyer *et al.* (2004) or by Pearce *et al.* (1999), although they studied the relationship between atopy and asthma *prevalence* in adults.

Carroll *et al.* (2006) studied a total of 400 children (7-18 years) with asthma in the UK (North Staffordshire and Sheffield). The authors concluded that increasing atopic sensitisation is associated with increasing disease *severity* in children with asthma.

Allergic sensitisation and family history of asthma as risk factors for asthma onset were also studied by Backlund *et al.* (2006). A cohort study followed 3525 Swedish children aged 7-8 years old in 1996, until they were 11-12 years old. The study found that sensitisation to any allergen (*Dermatophagoides Pteronyssinus*, *Dermatophagoides Farinae, Cladosporium* and *Alternaria*) was the strongest risk factor for current asthma (OR: 4.88, 95% CI 3.31-7.20) at age 7-8, increasing to OR 5.63 (95% CI 3.88-8.18) at age 11-12 with no difference between sexes. A family history of asthma was the second strongest risk factor with OR 3.04 (95% CI 2.07-4.47) at age 7-8 and OR 2.78 (95% CI 1.96-3.94) at age 11-12. The study also examined asthma remission, defined as subject reporting current asthma at one occasion, and reporting neither wheeze nor medication use in the past 12 months during the next occasion. Remission was found inversely correlated with allergic sensitisation. However, it should be mentioned that a study with a longer timescale should be carried out in order to confirm the figures on remission.

The studies described so far show that atopy plays a role in asthma persistence (prevalence), incidence and severity. There are significant geographic variations in asthma prevalence, but the strength of the association between asthma and atopy does not appear to vary significantly worldwide in adults. However, the population fraction

of asthma attributable to atopy varies in relation to the definition of asthma and of atopy. Furthermore, the population fraction of asthma attributable to dust mite sensitisation does appear to vary significantly worldwide – at least in adults - suggesting that the prevalence of sensitisation to dust mites (and exposure to dust mite allergen) is the main determinant of geographic variations in the population fraction of asthma attributable to atopy. In England and Wales the (adult) population fraction of (prevalent) asthma attributable to dust mite sensitisation varies from 19% to 36%. This means that 19-36% of adult asthma could be potentially prevented, if HDM sensitisation could be avoided. This raises the following questions: a) is there a dose-response relationship between HDM allergen exposure and sensitisation/asthma? b) are there threshold levels of HDM allergen exposure, below which sensitisation does not occur?.

The Committee on the Assessment of Asthma and Indoor Air, from the Institute of Medicine of the National Academy of Sciences (US) concluded that: "*There is sufficient evidence of a causal relationship between HDM allergen exposure and exacerbation of asthmatics specifically sensitized to dust mites. Continual exposure to dust mite allergens is also a contributing cause of chronic bronchial hyperreactivity. There is sufficient evidence of a causal relationship between dust mite allergen exposure and the development of asthma in susceptible children*" (National Academy of Sciences, 2000). The Committee specifies that 'causality' is not intended it in its old-fashioned concept of sufficient *and* necessary cause (*sufficient* cause: all persons exposed to x will develop asthma; *necessary* cause: all cases of asthma are caused by x). Rather, the Committee pointed out that it is generally recognized that most health outcomes of interest have multifactorial etiologies. Therefore, causality occurs if there is at least one person whose asthma was caused by a certain factor X.

Several studies support the notion that dust mites play a major role in asthma exacerbation and development in susceptible individuals. However, some authors are much more cautious (Pearce *et al.*, 2000). One of the issues under discussion is not necessarily whether exposure to dust mite allergen can "cause" asthma in at least one person, but rather whether HDM exposure causes asthma for a *significant* proportion in the asthmatics population. In the previous section it was highlighted that the population fraction of asthma attributable to dust mite sensitisation varies significantly worldwide – at least in adults - suggesting that the prevalence of

sensitisation to dust mites (and exposure to dust mite allergen) is the main determinant of geographic variations in the population fraction of asthma attributable to atopy (Sunyer *et al.*, 2004).

When considering the relationship between HDM allergens, atopy and asthma, a number of interrelated issues have to be considered:

1. Does exposure to HDM allergen increase the risk of *specific sensitisation* (in at risk individuals), and if so, is there a (linear) dose-response relationship between allergen exposure and sensitisation?
2. Does exposure to HDM allergen increase the risk of *asthma onset* (in at risk individuals?), and if so, is there a (linear) dose-response relationship between allergen exposure, HDM sensitisation and asthma onset?
3. Does exposure to HDM allergen increases the *severity of asthma symptoms* in sensitised individuals?
4. Can threshold levels for HDM allergen exposure be determined, above which the following occur: a) HDM sensitisation; b) asthma development; c) asthma exacerbation?

These issues are very important when trying to formulate strategies for: reducing symptoms in asthmatic patients, reducing the incidence of HDM sensitisation and reducing the incidence of asthma.

In 1989 a team of experts discussed the worldwide problem of dust mite allergens and asthma in an International Workshop under the auspices of the WHO (Platts-Mills and de Weck, 1989). One of the main conclusions of the workshop was the provisional recommendation of threshold levels for mite-allergen exposure. The experts proposed that a level of 2 μg of Der p1 per gram of dust (equivalent to 100 mites per gram) should be regarded as a risk factor for sensitisation and the development of asthma. The higher level of 10 μg of Der p1 per gram of dust (equivalent to 500 mites per gram) was proposed as a major risk factor for the development of acute asthma in mite-allergic individuals. In 1992, a second International Workshop took place, which confirmed the threshold levels and the dose-response relationship between HDM allergen exposure, HDM sensitisation and asthma development (Platts-Mills *et al.*, 1992). These recommended levels are often referred to in the literature as the "WHO threshold levels" for dust mite allergens and asthma.

However, since the formulation of the "WHO" threshold levels, further research has been carried out, which provides additional insight into the role of HDM allergen exposure on asthma, as well as into the exposure thresholds. Indeed, a *third* International Workshop of experts on dust mites and asthma took place in 1997, highlighting that the pattern of sensitisation to specific allergens reflects the *mean level* of allergen found in the houses of those communities where the patients live. The experts concluded that there was still sufficient evidence for a dose-response relationship between exposure to mite allergens and sensitisation to these allergens. They also highlighted that mite sensitisation is a major independent risk factor for asthma in New Zealand, coastal Australia, Florida, central Virginia. However, in other countries such as Scandinavia and the mountain states of the US, sensitisation to domestic animal allergens may have the strongest association with asthma. The experts therefore slightly changed their recommendations on threshold levels, concluding that in areas where the *mean level* of dust mite group I allergen in houses is 2 μg of Der p1 per gram of dust or more, sensitisation to mites has consistently been found to be associated with asthma. In the report of the third International Workshop there was no mention of the threshold of 10 μg of Der p1 per gram of dust. This is because the experts concluded that the relationship between HDM allergen exposure and asthma symptoms is complex, which makes the identification of a threshold level in HDM allergen exposure for asthma exacerbation quite difficult (Platts-Mills *et al.,* 1997).

Although a single threshold of HDM allergen exposure for exacerbation of asthma symptoms may be difficult to assess, some studies have correlated disease severity and allergen exposure. For example, Custovic *et al.* (1996) concluded that clinical activity and severity of asthma in mite-sensitive non-smoking adult patients is related to mite allergen exposure, with levels in beds being an important indicator that correlated with disease activity.

In identifying the threshold levels for mite sensitisation, the reports of the International Workshops did not explicitly make a distinction between 'at risk individuals' (i.e. with a family history of atopy and/or asthma) and 'not at risk individuals'. However, Custovic and Chapman highlighted that most studies which had investigated the relationship between exposure and sensitisation up to that time had focused on individuals at risk of developing atopy (Custovic and Chapman,

1998). Therefore, Custovic and Chapman concluded that exposure to 2 μg of Der p1 per gram of dust or more should be regarded as a risk factor for the development of mite-specific IgE antibody and asthma in *susceptible* children. This value is applicable to the population and should not be extrapolated to the clinical situation and to individual patients. Custovic and Chapman also concluded that a simple threshold level for provocation of asthmatic symptoms had not been yet identified. In a discussion paper, Marks also concluded that it seems likely that a threshold, below which sensitisation does not occur, is either much lower than 2 μg of Der p1 per gram of dust, or does not exists (Marks, 1998). However, Marks also concluded that there does appear to be an upper limit above which further increases in exposure do not cause any further increase in risk of sensitisation. Marks suggested that this upper limit is approximately 10 μg of Der p1 per gram of dust. In populations such as children in Sydney where virtually everyone is exposed to HDM levels > 10 μg of Der p1 per gram of dust, other factors influence the risk of developing allergy. Marks therefore concludes that HDM exposure is a necessary but not a sufficient factor in the development of HDM sensitisation.

The main conclusions emerging from reports such as those produced by the International Workshops on dust mites and asthma (Platts-Mills *et al.*, 1989, 1992, 1997) were based on the hypothesis that exposure to high levels of HDM allergen during early childhood contributes to HDM sensitisation and that continued exposure causes airway inflammation leading to the development of asthma in children. However, most studies carried out in the 1990s were either cross-sectional or case-control, where it is difficult to assess the role of HDM allergen exposure on asthma onset. A fundamental study on the role of HDM exposure and asthma onset in children was a *cohort* study of 67 British children at risk for allergic disease because of family history, where the relationship between Der p1 exposure and the *development* of sensitisation and asthma was investigated (Sporik *et al.*, 1990). The study concluded that there was a trend towards an increasing degree of sensitisation at the age of 11 with greater exposure at the age of 1 ($p=0.062$). Also, the relative risk of asthma development was 4.8 ($p=0.05$) for exposures to more than 10 μg of Der p1 per gram of dust.

The study by Sporik *et al.* has often been quoted in subsequent studies (included those produced by the WHO International Workshops) as proof of a causal relationship

between HDM exposure and childhood asthma onset. However, the study had some limitations: a fairly small sample size, the study focussed on at risk children (family history), and it was carried out in an area with relatively high levels of mite allergen. Subsequent cohort studies have presented a more contradictory picture on the role of HDM exposure in the onset of childhood asthma. In the German Multicentre Allergy Study, 939 newborn infants from 5 German cities were followed for 7 years (Lau *et al.*, 2000). The study was unable to find a consistent dose-response relationship for early indoor allergen exposure and "doctor's diagnosed asthma", "wheezing within the last 12 months", or "wheezing ever". However, sensitisation to mite and cat allergens was associated with indoor allergen exposure and with wheezing. The author of the study did highlight that the allergen levels found in carpets in their study were fairly low. Lau *et al.* conclude that whereas allergen exposure has a clear influence on atopy, in areas with moderate exposures the link to asthma is less pronounced.

A subsequent cohort study based in the UK established different conclusions from Sporik *et al.* (1990), and from Lau *et al.* (2000). Cullinan et al. (2004) followed 625 children in Ashford (Kent, UK) from birth to the age of 5.5 years, at which time 552 underwent skin prick testing for HDM and cat. When the impact of allergen exposure on sensitisation and on atopic wheeze was assessed (Fig. A.2.1), it was found that the exposure-response relationship for each allergen was neither linear nor monotonic, but showed an increase in risk at low levels of exposures, followed by a flattening or, in some cases, a reduction of risk at higher exposures. Different patterns were observed in relation to family history (atopic father) and birth order (first born).

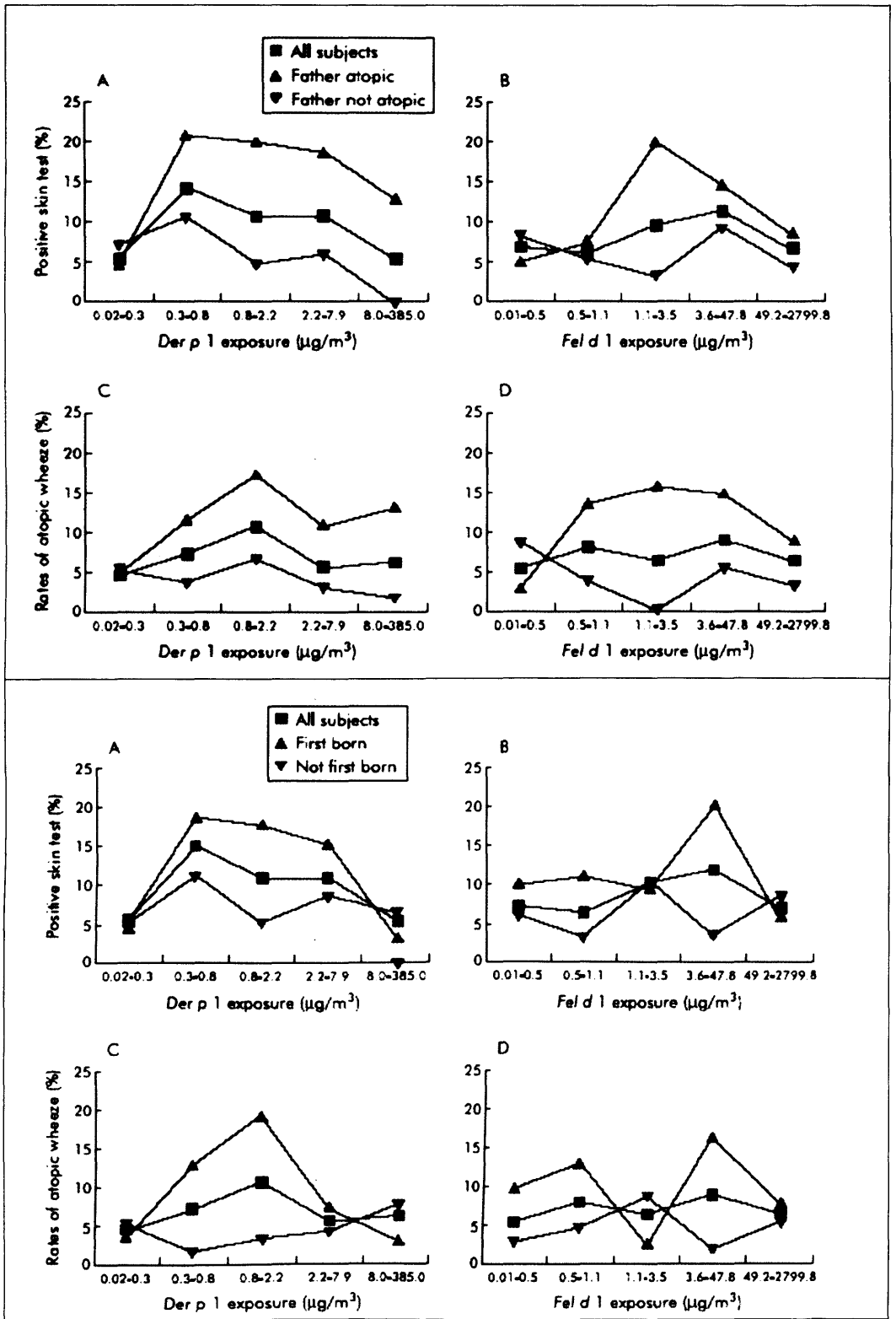


Fig A.2.1 Relationships between exposure to Der p1 or Fel d1, and sensitisation or atopic wheeze, in a cohort of British children (Cullinan *et al.*, 2004).

Another cohort study (Childhood Allergy Study) carried out in the US (Detroit) on 428 children from birth up to the age of 6 or 7 also confirmed that family history of atopy can be an effect-modifier in the exposure-response relationships between HDM allergen, atopy and asthma onset. They found that where absence of parental history of allergic disease appeared to decrease the risk HDM sensitisation at the age of 6 or 7, whereas the risk was increased in children with a parental history of allergic disease (Cole Johnson *et al.,* 2004).

The results obtained by Cole Johnson *et al.* were not reproduced by another cohort study carried out in the Netherlands (Brussee *et al.*, 2005). The Prevention and Incidence of Asthma and Mite Allergy (PIAMA) study investigated the effect of allergen exposure at 3 months of age on the development of sensitisation, wheeze, and physician diagnosed asthma in the first 4 years of life in a birth cohort of 3291 children. The results were stratified by maternal atopy. The results from PIAMA study on allergen exposure and specific sensitisation agree with the findings of a dose-response relationship found by the German study from Lau *et al.* (2000). However, both studies had low mite allergen levels. The PIAMA study results did not agree with those from the US Childhood Allergy Study (Cole Johnson *et al.*, 2004), where parental history was an effect-modifier. However, it should be emphasised that in the PIAMA study, dust samples were taken from the child's bed (which was new in 1/3 of the cases), while in the other studies - Lau *et al.*, 2000; Cullinan *et al.*, 2004; Cole Johnson *et al.*, 2004 - the samples were taken from carpets (living room for Cullinan *et al.*). The authors of the PIAMA study also highlighted that the cohort needed to be followed further, in order to assess the long-term consequences of early life exposures.

The issue of dose-response relationship between exposure to dust mite allergen and HDM sensitisation was also addressed by the PARSIFAL study, a cross-sectional study carried out in 5 European countries: Sweden, Switzerland, Germany, Austria and the Netherlands (Schram-Bijkerk *et al.*, 2006). The study included children aged 5-13 years, from a randomly selected population of 229 children from livestock farms, 122 Steiner children, with respectively 60 and 67 control children, with nearly equal numbers per country. The results showed that mite allergen levels were in the same order of magnitude for all groups of children. However, farm children had a lower prevalence of mite sensitisation. In the PARSIFAL study highest sensitisation rates

were observed in the intermediate exposure group, consistently across farm, Steiner and reference children. In order to study the dose-response relationship between HDM allergen exposure and HDM sensitisation, as well as the effect of microbial agents on such relationship, smoothed dose-response curves were determined by generalised cross validation. The curves were stratified by each microbial agent and dichotomised according to the median level of each agent (Figure A.2.2).

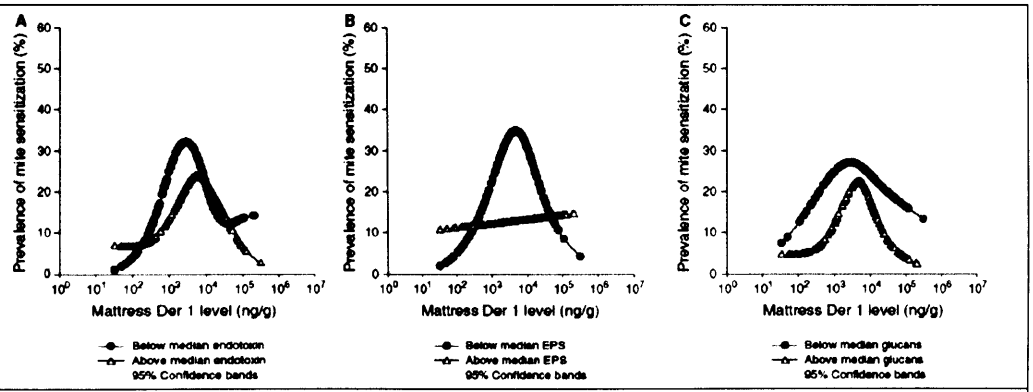


Fig A.2.2 Smoothed plots of the prevalence of mite sensitisation in relation to mite allergen levels, stratified by: A) dichotomised endotoxin levels; B) dichotomised extracellular polysaccharides (EPS) levels; C) dichotomised glucan levels in mattress dust (Schram-Bijkerk *et al.*, 2006).

Figure A.2.2 shows that for both endotoxin and glucan, the curve is bell-shaped below and above median levels. However, for EPS the curve is bell-shaped below median EPS levels but the curve is very much flattened above median EPS levels. The authors highlight that their results are not in line with previous cohort studies showing a linear dose-response relationship between mite allergen levels and mite sensitisation (e.g. Lau et al., 2000). The author of the PARSIFAL study suggest that this might be due to differences in allergen levels - which were rather high in their study – and/or in the study population. The results from the PARSIFAL study are in part similar to those found by Cullinan *et al.* (2004) in a UK cohort, where an increased risk of sensitisation was found at low allergen levels, and an attenuated risk was found at high levels. However, in Cullinan *et al.*, as well as in Cole Johnson *et al.* (2004), parental history of allergy appeared to be an effect-modifier for HDM allergen exposure and HDM sensitisation - although the relationship were not statistically significant - while this effect-modification was not observed in the PARSIFAL study.

The Asthma Multicenter Infant Cohort Study (AMICS) followed prospectively a representative population for 3 European centres: Ashford (Kent, UK), Menorca Island and Barcelona (Spain) (Torrent *et al.*, 2006). They aimed to assess the role of early exposure to Der p1 and Fel d1 in sensitisation at the age of 4 years. The results showed that the exposure profiles among centres were very different, with high Der p1 levels in Menorca and high Fel d1 levels in Ashford. However, the proportion of children sensitised to Der p1 did not differ greatly among centres. Figure A.2.3 and A.2.4 show the variations in sensitisation rates at the age of 4 years by Der p1 and Fel d1 levels in early infancy, after adjustment for confounding variables. The figure shows that Der p1 levels had a positive association with sensitisation for Barcelona only.

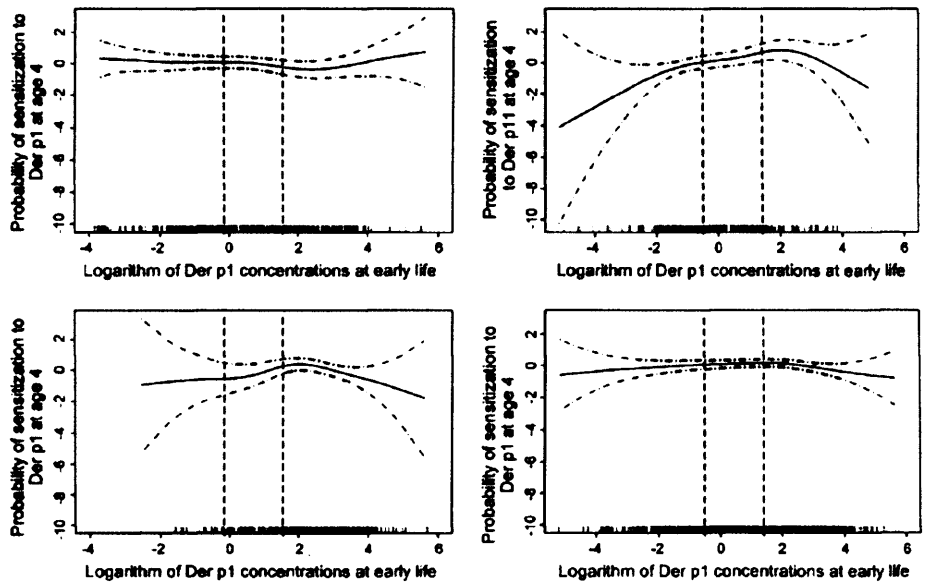


Fig A.2.3 Probability (logit scale) of sensitisation to house dust mite allergen (Der p1) at age 4 years and dust mite allergen exposure in early life. Upper left graph: *Ashford; upper right graph: Barcelona; lower left graph: Menorca; lower right graph: all centres.* Vertical line indicates tertile cut-off points. (Torrent *et al.*, 2006)

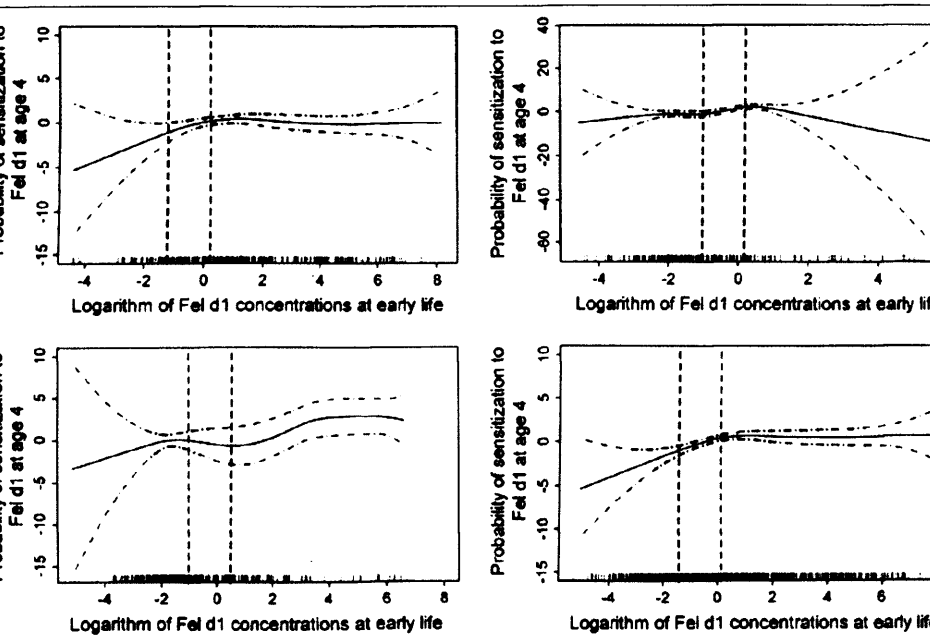


Fig A.2.4 Probability (logit scale) of sensitisation to cat allergen (Fel p1) at age 4 years and cat allergen exposure in early life. Upper left graph: *Ashford; upper right graph: Barcelona; lower left graph: Menorca; lower right graph: all centres.* Vertical line indicates tertile cut-off points. (Torrent *et al.*, 2006)

Pooled multiple regression models for Der p1 indicated that only maternal atopy and male sex had a statistically significant positive association with Der p1 sensitisation. There was no relationship with dust mite allergen exposure in early life and HDM sensitisation. A threshold level for sensitisation could not be found: even at a concentration of less than 0.032 μg Der p1/g of dust, 2 out of 20 children were sensitised. In summary, the authors of the AMICS study conclude that the dose-response relationships between allergen exposure and sensitisation differ between allergen and between geographical areas. The relationship between allergen exposure and sensitisation might not be linear.

This appendix discussed the role of house dust mites on HDM sensitisation, asthma development and asthma severity, with a focus on threshold levels of exposure. In summary, there is little doubt that exposure to HDM allergen leads to exacerbation of asthma symptoms in susceptible individuals, and that asthma severity is greater with greater exposure to HDM allergens. However, no single threshold level of HDM allergen exposure can be identified for exacerbation of asthma symptoms. Exposure to HDM allergen may lead to HDM sensitisation and to asthma development. However, there is some controversy on the extent of asthma onset which can be attributed to HDM exposure. Furthermore, there is some contradictory evidence on the relationship between exposure to HDM allergens, and HDM sensitisation or asthma onset. This is mostly because these relationships can vary in relation to: study population (i.e. genetic factors and typical exposure levels), family history of asthma/atopy, and other confounding factors – particularly the presence of potentially "protective" factors in the environment, such as endotoxins or extracellular polysaccharides (EPS). Because of these factors, it is unwise to identify a threshold level of HDM exposure for sensitisation or asthma onset, which can be applied to any population. Some evidence suggests that in England and Wales the (adult) population fraction of (prevalent) asthma attributable to dust mite sensitisation varies from 19% to 36%. Some evidence also suggests that in some study populations HDM sensitisation might occur at intermediate exposure levels, with a bell-shaped relationship (possibly modified by other potentially "protective" factors). This should be taken into account in any primary prevention studies aiming at reducing HDM allergen levels for the reduction of HDM sensitisation.

Appendix A.2: References

Backlund AB, Perzanowski MS, Platts-Mills T, Sandstrom T, Lundback B, Ronmark E, (2006), Asthma during the primary school ages - prevalence, remission and the impact of allergic sensitization, *Allergy*, 61(5): 549-55.

Brussee JE, Smit HA, Van Strien RT, Corver K, Kerkhof M, Wijga AH, Aalberse RC, Postma D, Gerritsen J, Grobbee DE, De Jongste JC, Brunekreef B, (2005), Allergen exposure in infancy and the development of sensitization, wheeze, and asthma at 4 years, *The Journal Of Allergy And Clinical Immunology*, 115 (5): 946-952.

Carroll WD, Lenney W, Child F, Strange FC, Jones PW, Whyte MK, Primhak RA, and Fryer AA, (2006), Asthma severity and atopy: how clear is the relationship?, *Archives of Disease in Childhood*, 91:405-409.

Cole Johnson C, Ownby DR, Zoratti EM, Alford SH, Williams KL and Joseph CLM, (2002), Environmental epidemiology of pediatric asthma and allergy, *Epidemiologic Reviews*, 24: 154-175.

Cullinan P, MacNeill SJ, Harris JM, Moffat S, White C, Mills P and Newman Taylor AJ, (2004), Early allergen exposure, skin prick responses, and atopic wheeze at age 5 in English children: a cohort study, *Thorax*, 59:855-861.

Custovic A and Chapman M, (1998), Risk levels for mite allergens. Are they meaningful?, *Allergy*, 53(Suppl. 48):71-76.

Custovic A, Taggart SC, Francis HC, Chapman MD, Woodcock A, (1996), Exposure to house dust mite allergens and the clinical activity of asthma, *Journal of Allergy and Clinical Immunology*, 98(1): 64-72.

Jaakkola MS, Ieromnimon A, Jaakkola JJK, (2006), Are atopy and specific IgE to mites and molds important for adult asthma?, *Journal of Allergy and Clinical Immunology*, 117 (3): 642-648.

Jarvis D, Burney P, (1998), The epidemiology of allergic disease, *BMJ*, 316: 607-610.

Lau S, Illi S, Sommerfeld C, Niggemann B, Bergmann R, von Mutius E, Wahn U, and the Multicentre Allergy Study Group, (2000), Early exposure to house-dust mite

and cat allergens and development of childhood asthma: a cohort study, *The Lancet*, 356(9239): 1392-1397.

Marks GB, (1998), House dust mite exposure as a risk factor for asthma: benefits of avoidance, *Allergy*, 53(Suppl. 48): 108-114.

National Academy of Sciences (Committee on the Assessment of Asthma and Indoor Air, Institute of Medicine, U.S.), (2000), *Clearing the Air,* Washington: National Academy Press.

Pearce N, Douwes J, Beasley R., (2000), Is allergen exposure the major primary cause of asthma?, *Thorax*, 55(5): 424-31.

Pearce N, Pekkanen J, and Beasley R, (1999), How much asthma is really attributable to atopy?, *Thorax*, 54: 268–272.

Platts-Mills T and de Weck A, (chairmen), (1989), Dust mite allergens and asthma – a worldwide problem. Report of an International Workshop, *Journal of Allergy and Clinical Immunology*, 83(2, part 1): 416-427.

Platts-Mills TA, Thomas WR, Aalberse RC, Vervloet D, Chapman MD, (1992), Dust mite allergens and asthma: report of a second international workshop, *Journal of Allergy and Clinical Immunology*, 89(5): 1046-60.

Platts-Mills TA, Vervloet D, Thomas WR, Aalberse RC, Chapman MD, (1997), Indoor allergens and asthma: report of the Third International Workshop, *Journal of Allergy and Clinical Immunology*, 100(6 Pt 1): S2-S24.

Rockhill B, Newman B, Weinberg C, (1998), Use and Misuse of Population Attributable Fraction, *American Journal of Public Health*, 88(1): 15-19.

Schram-Bijkerk D, Doekes G, Boeve M, Douwes J, Riedler J, Üblagger E, von Mutius E, Budde J, Pershagen G, van Hage M, Wickman M, Braun-Fahrländer C, Waser M, Brunekreef B, the PARSIFAL study group, (2006), Nonlinear relations between house dust mite allergen levels and mite sensitization in farm and nonfarm children, *Allergy*, 61(5): 640-647.

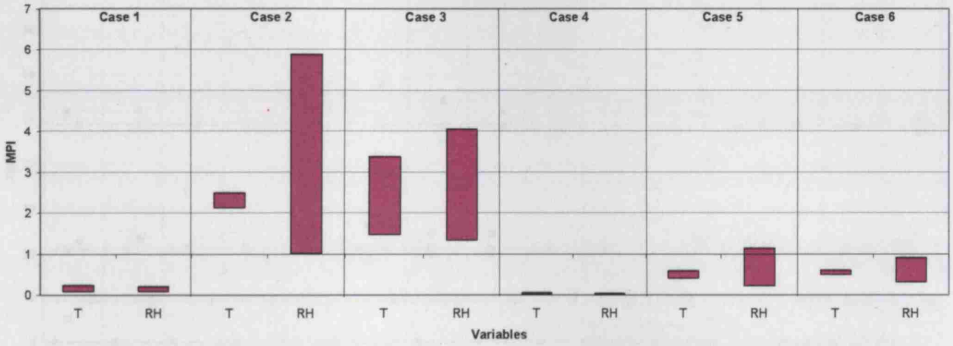
Sporik R, Holgate ST, Platts-Mills TA, and Cogswell JJ, (1990), Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. A prospective study, *The New England Journal of Medicine*, 323(9): 502-507.

Sunyer J, Jarvis D, Pekkanen J, Chinn S, Janson C, Leynaert B, Luczynska C, Garcia-Esteban R, Burney P, Anto JM; European Community Respiratory Health Survey Study Group, (2004), Geographic variations in the effect of atopy on asthma in the European Community Respiratory Health Study, *Journal of Allergy and Clinical Immunology*, 114(5): 1033-1039.

Torrent M, Sunyer J, Muñoz L, Cullinan P, Iturriaga MV, Figueroa C, Vall O, Taylor AN and Anto JM, (2006), Early-life domestic aeroallergen exposure and IgE sensitization at age 4 years, *Journal of Allergy and Clinical Immunology*, 118(3): 742-748.

Appendix to Chapter 9

A.9: Sensitivity Analysis, Further Graphs



A.9.1

Sensitivity of the MPI model to +/- 1-% changes in temperature and RH

Following is a number of graphs illustrating the sensitivity of the Popmite model to changes in input variables. It may be helpful to illustrate one of the graphs. For example, in Figure A.9.2 the parameters which have been independently assessed for the sensitivity analysis are on the x-axis (abbreviations for each parameter are described in Table 9.4.1, chapter 9). Each parameter is separated by dashed vertical lines. The Popmite predictions are given on the y-axis, representing the predicted final *adult* population. Each data series represents one hygrothermal input base-case (as summarised in Table 9.4.2, chapter 9). For example, the green squares correspond to the "Average" input hygrothermal base-case. The green horizontal line corresponds to the base-case prediction (nearly 50 mites). The green triangles above and below this line represent the variation in predictions, due to changes in the x-axis input parameters (+/- 10%). For example, a change in input temperature (first variable on the x-axis) results in a *reduction* in predictions (both triangles *below* the solid green line), regardless of whether such change was an increase or a decrease from base-case temperature. On the other hand, changes in other variables do not result in changes in predictions (e.g. input variable "Fast Up"). Figure A.9.3 and A.9.4 are similar to Figure A.9.2, except that they show Popmite predictions for juveniles and for eggs, respectively.

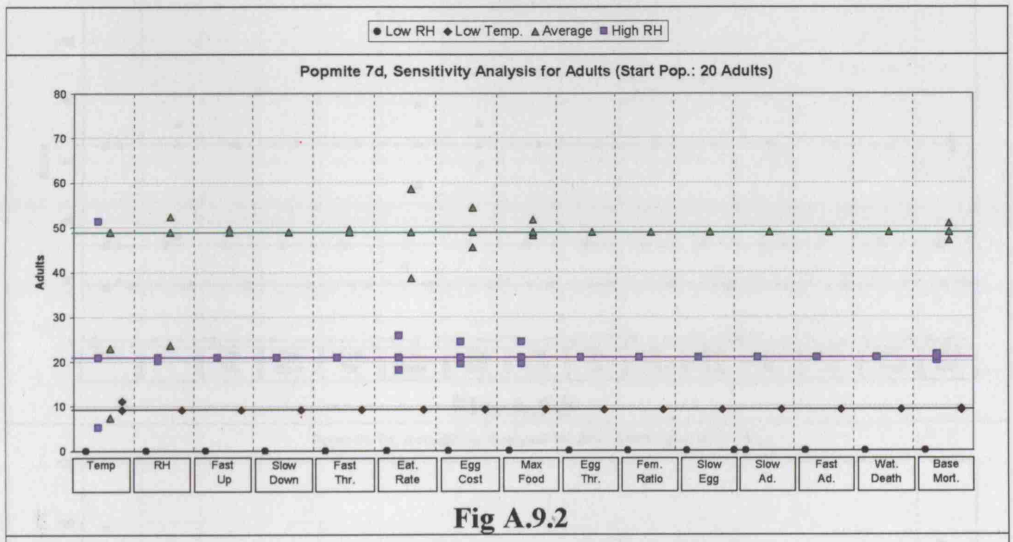


Fig A.9.2

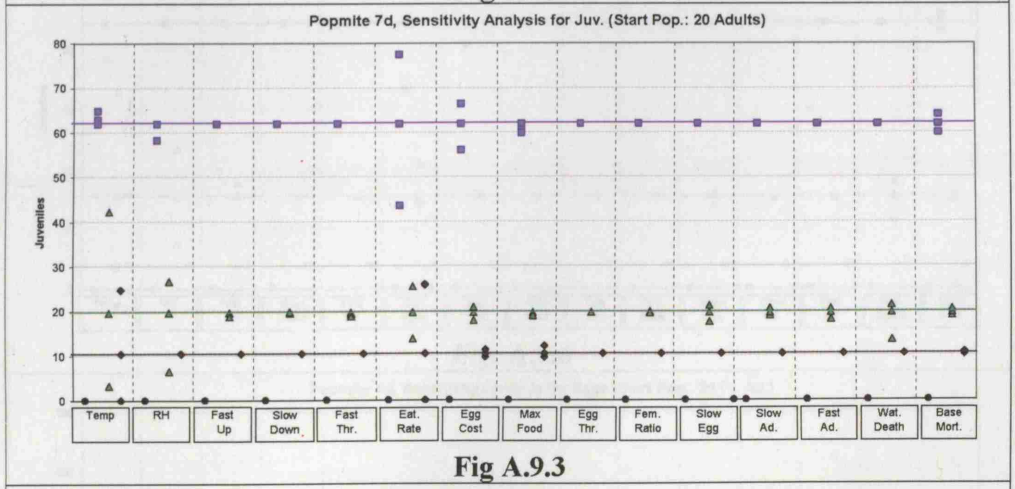


Fig A.9.3

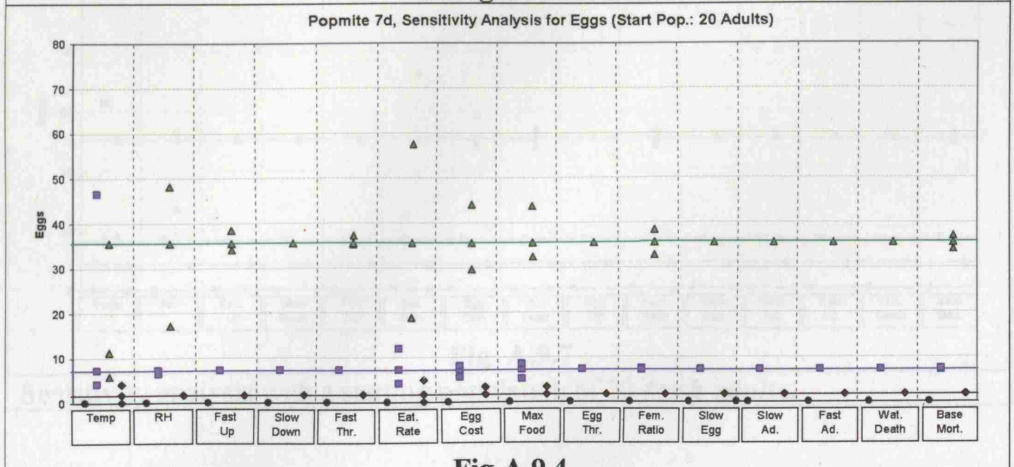
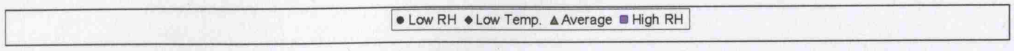


Fig A.9.4

Sensitivity analysis for Popmite, with a starting population of 20 adults.



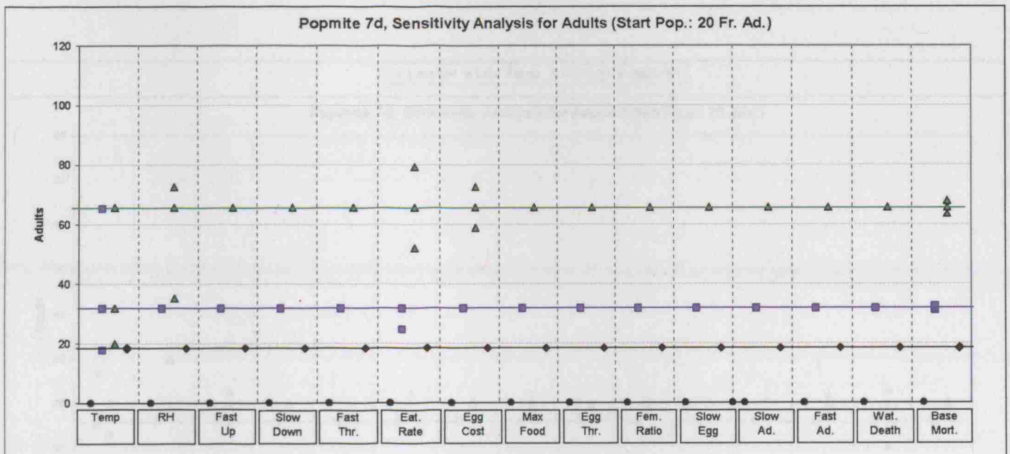


Fig. A.9.5

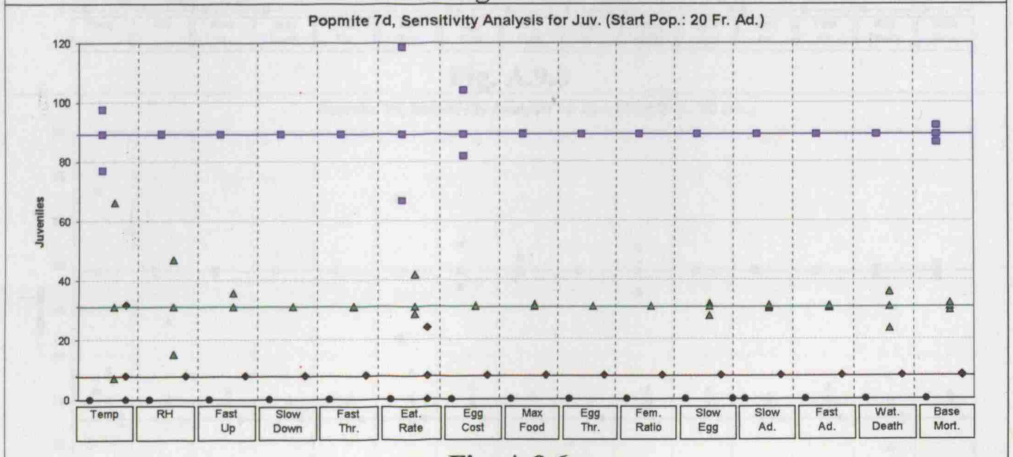


Fig. A.9.6

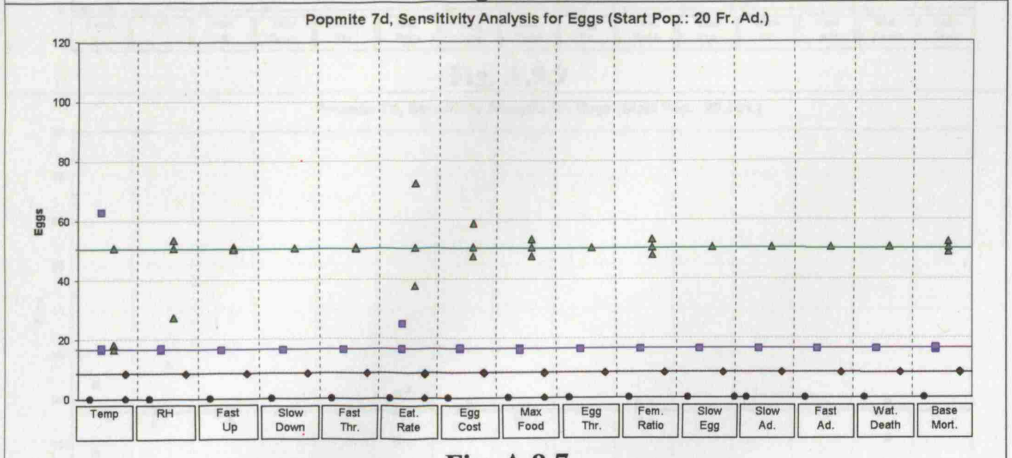


Fig. A.9.7

Sensitivity analysis with a starting population of **20 *fresh* adults.**

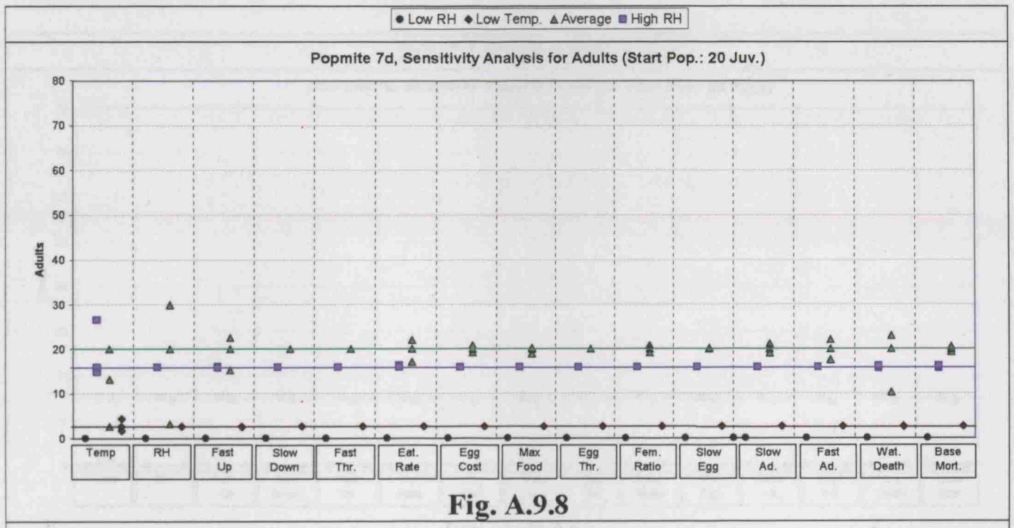


Fig. A.9.8

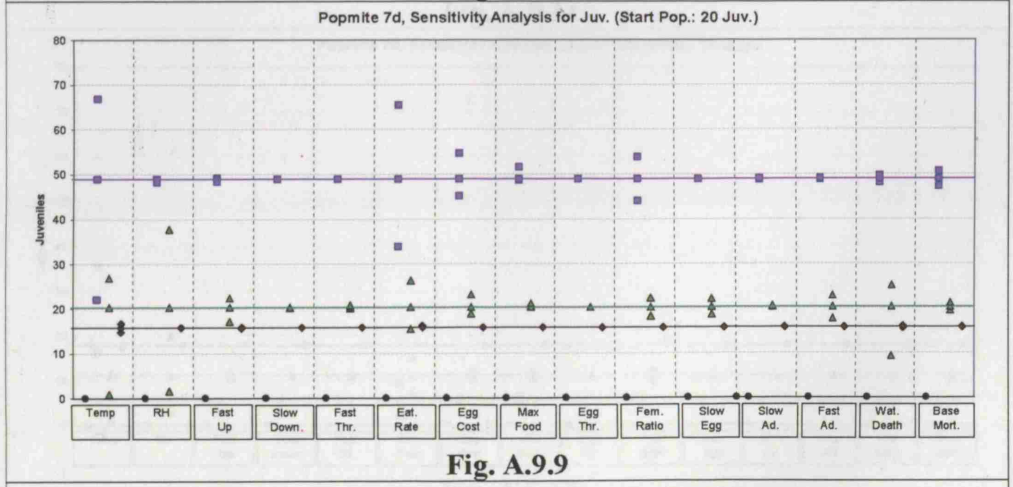


Fig. A.9.9

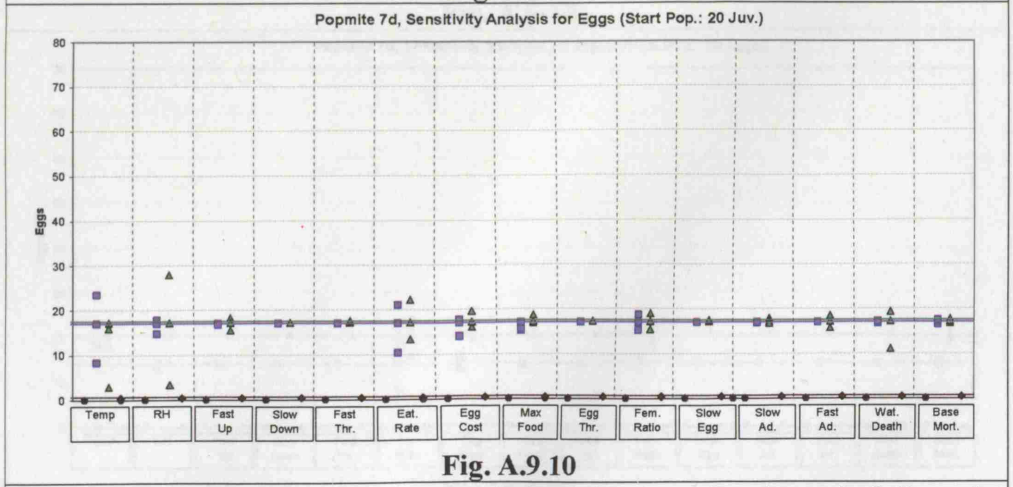


Fig. A.9.10

Sensitivity analysis with a starting population of **20 juveniles** (spread of all ages).

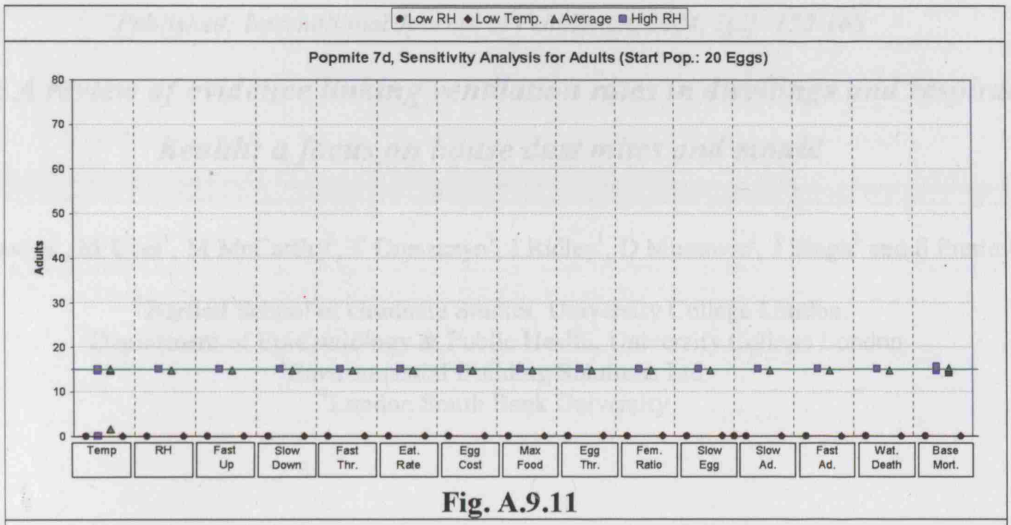


Fig. A.9.11

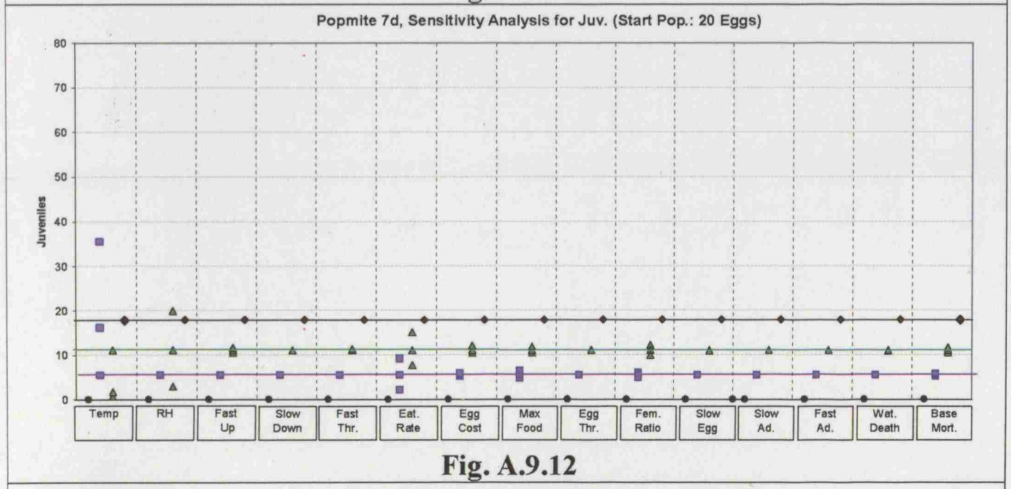


Fig. A.9.12

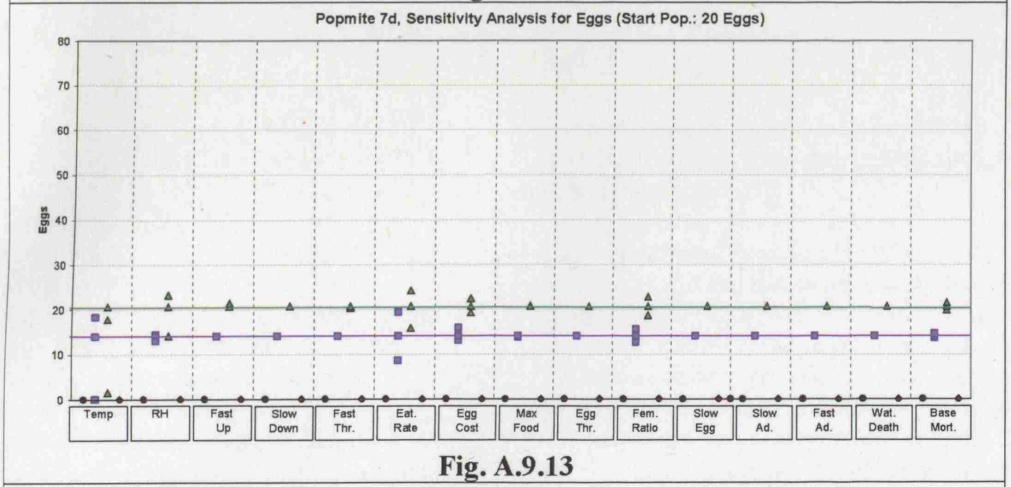


Fig. A.9.13

Sensitivity analysis with a starting population of **20 eggs** (spread of all ages).

Appendix A.0: Published Papers

Published: International Journal of Ventilation, 2004, 3(2): 155-168

A.0.1: *A review of evidence linking ventilation rates in dwellings and respiratory health: a focus on house dust mites and mould*

M Davies[1], **M Ucci**[1], M McCarthy[2], T Oreszczyn[1], I Ridley[1], D Mumovic[1], J Singh[3] and S Pretlove[4]

[1]Bartlett School of Graduate Studies, University College London.
[2]Department of Epidemiology & Public Health, University College London
[3]Environmental Building Solutions Ltd.
[4]London South Bank University

Abstract

This paper reviews the literature for evidence of links between ventilation rates in dwellings and moisture related respiratory health with a particular focus on house dust mites (HDM) and fungal growth. There is general consensus that a link exists between ventilation rates in dwellings and respiratory hazards (for example HDM). There is also general consensus of a link between these respiratory hazards and respiratory problems, but it is not clear to what extent hazards cause ill-health. Most existing data are inadequate for conclusions to be drawn whether ventilation rates directly cause respiratory problems. We discuss the many difficulties in attempting to establish these relationships, and suggest the need for larger studies.

Key words: review, ventilation rates, respiratory health, house dust mites, mould.

1. Introduction

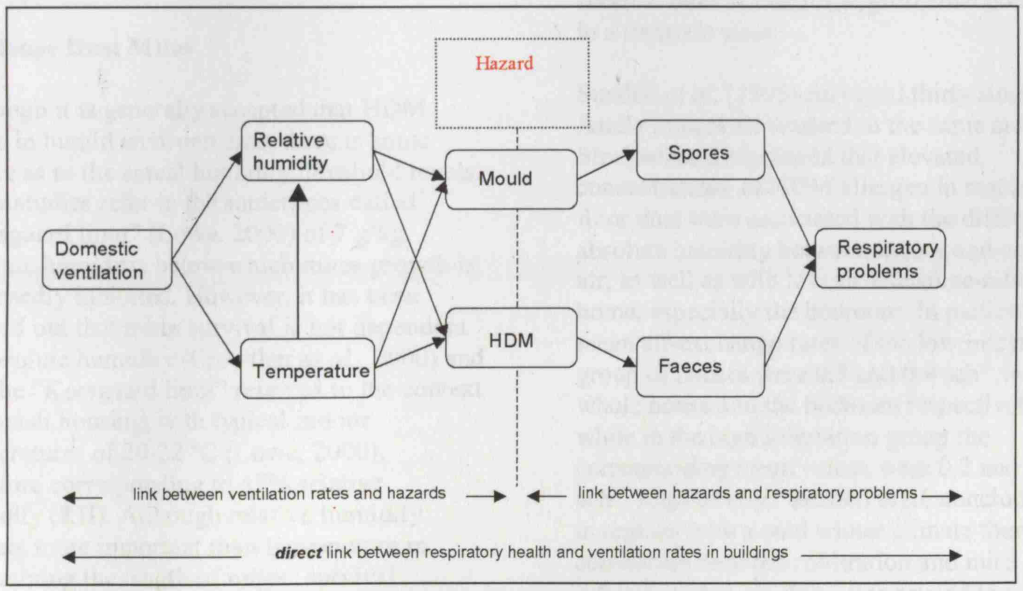


Figure 1. Postulated pathways between ventilation and related respiratory problems

This review focuses on *moisture related* respiratory hazards (in this case HDM and fungal growth). In a most basic manner, figure 1 shows the postulated pathways between ventilation and relevant moisture related respiratory problems (note that this diagram only shows a small element of the total complex system). Convenient sub-categories of links are also shown. The review will deal with these links in terms of publications which relate to:

- a link between ventilation rates in dwellings and moisture related respiratory hazards
- a link between moisture related respiratory hazards and respiratory problems
- a *direct* link between moisture related respiratory health and ventilation rates in buildings

The following three sections examine the evidence currently available in the literature for these links.

Part of the complexity involved in research in this area is that in most cases it is difficult to measure ventilation rates in a meaningful and accurate manner for the number of properties that are required to provide any statistically significant health data. Also, most changes to ventilation rate have occurred at the same time as other changes e.g. other improvements to the building fabric. Frustratingly, it is not easy to infer the ventilation rate of a property from other building factors such as the age of a property or occurrence of draught stripping. In addition, there are theoretical mechanisms which mean

that increased air ventilation is not always beneficial to health (e.g. indoor generated pollutants may decrease but externally generated pollutants may increase).

2. Studies relating to a link between ventilation rates in dwellings and moisture related respiratory hazards

There are two key moisture related respiratory hazards – house dust mites and mould growth. The following two subsections review the environmental conditions required for house dust mites and mould and the link with ventilation.

2.1 House Dust Mites

Although it is generally accepted that HDM thrive in humid environments, there is some debate as to the actual humidity threshold levels. Many studies refer to the sometimes called "Korsgaard limit" (Lowe, 2000) of 7 g/kg absolute humidity, below which mites growth is supposedly inhibited. However, it has been pointed out that mites survival is not dependent on absolute humidity (Crowther *et al.*, 2000) and that the "Korsgaard limit" referred to the context of Danish housing, with typical indoor temperatures of 20-22 °C (Lowe, 2000), therefore corresponding to 45% relative humidity (RH). Although relative humidity appears more important than temperature in determining the length of mites' survival (Crowther *et al*, 2000), the Critical Equilibrium Humidity (CEH) – below which mites dehydration occurs – appears to be dependent on temperature as well (Cunningham, 1996; Arlian and Platts-Mills, 2001). In establishing a limit for the psychrometric control of the mite species *D. farina* Cunningham (1996) suggested that indoor relative humidity should be kept below 40% at 16 °C, 45% at 21 °C and 50% at 26 °C. On the other hand, Raw (2001) states that RH at 35-40% in winter, although difficult to achieve in the UK, should be adequate to prevent mite proliferation. Temperature is also an important factor for the egg to adult time span.

It is important to note that HDM survival is not only dependent upon microclimatic hygrothermal conditions but also upon the length of time for which such hygrothermal conditions occur. For example, some studies suggest that adult mites die of dehydration in 5 to 11 days, depending on temperature (25 °C - 34 °C) when continuously exposed to RHs of 40% or 50% (Arlian and Platts-Mills, 2001). It should also be pointed out that mites are able to move to areas where more favourable environmental conditions occur. Furthermore, some differences have been found between laboratory and 'wild' HDM populations (Crowther *et al.*, 2000). However, most research studies on mites' survival ability have been conducted under steady-state laboratory conditions using cultured populations of HDMs. Although much has been established on HDMs' physiology, further research is required providing a complete knowledge on wild mites' survival rates at various hygrothemal conditions in a transient state.

Sundell *et al.* (1995) surveyed thirty single-family houses all situated in the same area of Stockholm. They found that elevated concentrations of HDM allergen in mattress and floor dust were associated with the difference in absolute humidity between indoor and outdoor air, as well as with low air-exchange-rates of the home, especially the bedroom. In particular, the mean air-exchange-rates of the low infestation group of houses were 0.3 and 0.9 ach^{-1} for the whole house and the bedroom respectively, while in the high infestation group the corresponding mean values were 0.2 and 0.2 ach^{-1} respectively. Sundell *et al.* concluded that in regions with a cold winter climate there is a correlation between infiltration and mite infestation, but air-flow rates related to number of people in the home appears a stronger indicator of HDM infestation than air-flow rates related to home volumes.

Several studies have been carried out where mechanical ventilation with heat recovery (MVHR) and/or dehumidifiers were utilised in order to reduce relative indoor humidity levels in winter and consequently mite concentrations. However, although the use of MVHR appears to have proven quite successful in Scandinavian countries (e.g.: Emenius *et al.*, 1998; Harving *et al.*, 1994), such an approach has caused some conflicting results in the UK. A study (Fletcher *et al.*, 1996) conducted in the North-West of England on 18 houses – 9 with MVHR and 9 control houses – concluded that "the MVHR unit does not reduce indoor humidity to levels capable of retarding mite population growth and decreasing mite allergens in the type of houses

predominantly found in the mild and humid climate of the North-West of England" (*ibid.*, p.1051).

In a later study (Niven *et al.*, 1999), an additional central dehumidification modification of the MVHR (MVHRcd) was adopted in order to further assess the viability of the psychrometric control of HDM in the UK. Ten active houses were fitted with adapted MVHRcd units; relative humidity and allergen levels were monitored for 15 months and compared with the correspondent results of 10 control houses. The target temperature and humidity for the bedrooms in the active houses were 45% RH or 7 g/kg absolute humidity at 21 °C. The researchers concluded that "the MVHRcd system failed to confer a benefit in terms of mite allergen reduction" (*ibid.*, p.756). However, the authors also pointed out that the buildings' air-tightness might have compromised the effectiveness of the MVHRcd system. No measurements of air-infiltration were carried out to determine is this was a possible explanantion.

In a study carried out by Berry *et al* (1996), it was shown that the houses which rarely or never opened windows had higher mean yearly average mite counts taken from the bedroom carpet. This was not observed with counts obtained from the living room carpet, when the degree of opening the living room window was examined.

Several studies report that HDM numbers increased with the severity of the dampness in the property (Adan *et al* 1988 and Hart and Whitehead 1990). Toma *et al* (1993) found that ventilation did not influence mite numbers, but Korsgaard (1979) found that there was a tendency for higher numbers of mites in houses where the number of airing hours (opening up windows) was low. Korsgaard (1979) also found that there was no correlation between the temperature and mite counts. However, Irie *et al* 1990 found a link between increasing mite numbers and increasing indoor temperatures.

Modeling can provide an insight into this issue. As a result of a two-year research project funded by EPSRC in the UK, two models have been developed for the prediction of HDM in houses (Crowther *et al.*, 2002). The model 'BED3' has a steady-state hygrothermal model linked to an empirical population model. For the latter, laboratory measurements were undertaken with populations of HDM kept for three weeks at different combinations of steady RH and temperature, covering the range of conditions typical of UK dwellings. The more complex model 'Lectus' is a transient, three-dimensional model that simulates all stages of mite development. The population model adopted in Lectus is based on published data for the *Dermatophagoides pteronyssinus*, the most common mite species in the UK. With regard to the Lectus population model, the authors of the research pointed out that the existing published data are not complete but there was sufficient information, making simple assumptions, to derive curve-fitted equations. The models BED3 and Lectus were used to examine a range of issues, including the effect of ventilation rates on HDM populations. The study concluded that:

- Small reductions in ventilation rate below 0.5 ach^{-1} can have a dramatic impact. Modelling suggests that reducing from 0.5 to 0.4 ach^{-1} can increase the mite population by 100 times. However, an increase to above 0.7 ach^{-1} can also lead to an increase in the mite population in a fuel poor dwelling
- Raising bedroom temperatures from 16°C to 18°C, i.e. without reducing ventilation, can result in a significant (factor of ten) reduction in mite numbers. The increase in bedroom temperatures over the last 50 years, partly as a result of increased central heating and improved insulation, is therefore likely to have had beneficial effects. This supports the case for continuing to improve the UK's housing stock.
- Modelling suggests that building occupant density is a key parameter in determining house dust mite populations. Increasing the number of occupants in a dwelling from 4 to 6 can increase the mite population by 10,000 due to the increased moisture production in the property.

The role of indoor temperatures in the correlation between air-leakage and HDM population is also highlighted by Lowe (2000). The author considered the Critical Equilibrium Humidity (CEH) defined by Cunningham (Cunningham, 1996) of 40% RH at 16 °C and 45% at 21 °C. Modelling based on a typical UK dwelling and Kew weather data showed that at low internal temperatures the "Cunningham limit" is exceeded for most of the winter and increasing the ventilation rate does not improve

the situation greatly. At high internal temperatures, problems appear likely to occur only at ventilation rates significantly less than 0.5 ach^{-1}. Ridley *et al.* (2003) also found through modelling that an air-infiltration rate below 0.5 ach leads to an indoor RH greater than 70% in fuel rich dwellings (0.7 ach^{-1} for fuel poor dwellings). While in fuel rich dwellings the RH rapidly decreases with an increase in air-leakage, in fuel poor dwellings (who cannot afford to maintain always comfortable indoor temperatures) such inverse correlation does not occur.

2.2 Mould

Woolliscroft (1997) stated that the high level of condensation and mould in the UK is the consequence of the small size of the dwellings, low temperatures, high absolute humidity of the incoming air, and high occupancy of dwellings. 35% of dwellings were affected by condensation and 17% by mould growth. Comparing these results which were based on the English House Condition Survey 1988, with the same survey in 1996, it can be noticed that the incidence of mould growth of any severity has fallen to 14.6% of the total stock (DETR, 1996). The latest house condition survey published in 2001 omitted the collection of any condensation and mould data (ODPM, 2003).

A study by the UK Building Research Establishment (BRE) (Research project number EP228, 1990) revealed that recently built one bedroom and bedsit homes in the UK had significant condensation problems, which could lead to mould growth and proliferation. This study gave an indication of the factors related to condensation for example ventilation, air movement, heating and insulation. Their study indicated that the factors such as ventilation, air movement, heating and insulation were more important than occupant behaviour and energy consciousness and the most important occupant characteristics were the number and age of occupants (Raw and Fox 1990 in BRE study, Research project number EP228). BRE (Hunter *et al* 1988 and 1996) carried out biological assessments of houses and their investigation revealed that the most influential factor affecting the fungal counts appeared to be season.

In summary, it would appear that there is general consensus that links do exist between ventilation rates and moisture related respiratory hazards. We will now move on to consider the links between these hazards and possible respiratory problems.

3. Studies relating to a link between moisture related respiratory hazards and respiratory problems.

The most commonly perceived health effect arising from exposure to airborne moulds and other microorganisms, for example HDM, is allergy. Allergy-related diseases include asthma, rhinitis, and eczema or the less common diseases of extrinsic allergic alveolitis (hypersensitivity pneumonitis) and allergic bronchopulmonary aspergillosis (Pope *et al* 1993). Again, this section initially discusses the link between mites and health before moving on to consider mould.

3.1 House Dust Mites

HDM allergens are mostly present in their faecal pellets and they can trigger Type I allergic reactions, including asthma. Some studies also suggest that HDM allergens are associated with other health problems such as eczema and perennial allergic rhinitis. The evidence is reviewed by Raw, (Raw, 2001) who reports that "levels of mite allergen in the dust in most UK homes are high enough to cause sensitisation and it is possible that most people in the UK are exposed to enough mite allergen to cause asthma if they are susceptible to this disease for genetic or other reasons" (*ibid.*, 2001, p.15). However, the contribution of HDM as a direct cause of asthma, in comparison with many other predisposing and precipitating causes, is not known

Generally, the effects of pollutants on the lung can be categorised as irritation, inflammation, bronchoconstriction and sensitisation. Some of the more potent agents of allergic lung disease are found in indoor environments; such aeroallergens (house dust mite and moulds) have been recognised for many years. Inner city children have the highest prevalence and the highest mortality rates for asthma in the USA (Call *et al* 1992) and these children also have a high prevalence of dust mite sensitisation (Platts-Mills and Weck, 1989). However, many other factors could also contribute to this relationship

House dust mites, moulds and, less commonly, amoebae can colonise building structures, services, furnishing and finishes (e.g. Singh, 1999). House dust mites, fungi and yeasts are potent sensitizers, and they flourish in an environment of high relative humidity and low ventilation. Fragments of these organisms or their decayed material or their metabolites, becoming airborne, can be inhaled and cause allergic disease.

An important meta analysis of 23 patient-level intervention studies (Goetzsche *et al* 1998) was derived from 229 reviewed papers. A total of 230 patients, all showing mite sensitivity shown by skin-prick, were divided between intervention cases and non-intervention controls. Interventions were: 6 using chemical methods, 13 physical methods and 4 combination. No statistical difference was found in symptom responses between intervention and controls. The authors consider the sample size of strong statistical power and so the 'most likely explanation' given is either insufficient reduction in house mite levels or other allergens coexisting. In a dissenting editorial, Strachan (1998) suggested that sub-group analysis could still reveal clinically useful interventions with larger studies.

3.2 Mould

Now turning to mould in more detail, a number of studies have shown that mould growth in damp housing was associated with childhood respiratory illness; wheezing and asthma (Su *et al* 1990; Flannigan *et al* 1990; Dales *et al* 1990; White 1990; Spengler et al 1993; Husman *et al* 1993). None of these studies was in a peer-review health journal, and do not necessarily demonstrate a causal relationship

The effect of damp and mould in the home on respiratory health was reviewed by Peat *et. al.* (1998). The reviewer suggested that houses need to be specifically designed for primary prevention of respiratory problems associated with indoor allergen proliferation rather than using post hoc procedures to improve indoor climate and reduce allergen load as a secondary or tertiary preventive strategy. It was strongly emphasised that studies with large sample sizes were needed to measure whether intermittent peak exposures or low cumulative exposures to indoor allergen pose a clinically important risk.

Mould in damp buildings has recently emerged as an indoor environmental hazard of some concern (Lange et al 1993, Singh 1994a and Rylander 2003), although the issue has existed for centuries (Rautuiala *et al* 1998). A large volume of literature is appearing in journals particularly related to characteristics, distribution, public health, exposure, and health relationships for microbes, including fungi, and the indoor environment (Kalliokoski 2003, Lugauskas *et al* 2003, Adeeb and Shooter 2003,Sarca *et al* 2002, Menetrez *et al* 2002, Menetrez and Forde 2002, Kemp *et al* 2002a, Kemp *et al* 2002b and Mussalo-Rauhamaa *et al* 2003).

There are more than 100,000 species of fungi, and the genera and species possibly linked to human disease involve a wide array of both common and rare moulds. Fungi produce large numbers of spores and when these spores are liberated from infected buildings to the indoor air, they can be regarded as organic dust. These spores can, like other types of dust, sediment on surfaces or can be inhaled by occupants and deposited on the mucosal surface of the upper airways and in the eyes.

Microorganisms and their metabolites may cause a range of respiratory symptoms, depending upon the species, the exposure and the immune status of the subject (Singh, 1994a; Singh 1994b; Lacey, 1994; Comtois and Garcia, 1994).

Garret *et al* (1998) reported that no significant association between total viable mould concentrations and health outcomes was seen despite significant associations with specific genera. Reporting on the findings of the PEACE study, Andriessen *et al* (1998) concluded that Peak Flow (PEF) variability in atopic children was associated with (but not necessarily caused by) reported moulds in the home.

A few governmental agencies have published guidelines on mould assessment and remediation but most are very general in nature (Minnesota Department of Health. 2001and New York City Department of Health 2000 and Rao *et al* 1996). Some guidelines focus on toxogenic moulds, including Stachybotrys chartarum, which have

been reported in association with health conditions including acute pulmonary haemorrhage (Chapman 2003).

It is helpful to conclude this section of the review with the findings of a recent report (Raw *et al,* 2001). This study placed HDM in the highest level risk group with regard to health and safety hazards in homes. Fungal growth was placed in the second highest level risk group. In this study, hazards were placed in rank order, from the perspective of deciding whether preventative action was needed. It appears that the actual health risk from mould in buildings has yet to be quantified e.g. Chapman *et al* (2003) and Bornehag *et al* (2004). While repeated exposure to large amount of fungal propagules risks the development of specific allergic reactions, there is no adequate evidence of serious health hazards caused by so-called 'toxic' moulds.

In summary, there appears to be general consensus that a link exists between HDM and mould (i.e. respiratory hazards) and respiratory problems Having previously indicated that there is also general consensus that links do exist between ventilation rates and these respiratory hazards we now conclude by exploring what evidence exists to support a *direct* link between ventilation rates and respiratory problems.

4. Studies relating to a *direct* link between moisture related respiratory health and ventilation rates

The EUROVEN group recently reviewed the scientific literature relating to the effects of ventilation on health, comfort and productivity in non-industrial environments (Wargocki *et al* 2002). The study concluded that there was a strong association between ventilation and health. Studies judged conclusive implied that low ventilation rates in homes may be one of the factors exacerbating allergies due to the increased rate of infestation of HDM. The study also noted however, that more information is required on links between ventilation rates and health in homes.

A recent study (Emenius *et al* 2004) was undertaken to examine the impact of building characteristics and indoor air quality on recurrent wheezing in infants. The study found that whilst building-related exposures appear to have a major impact on children's health, this was not primarily explained by differences in ventilation systems, air change rate or HDM infestation.

In some UK studies the adoption of MVHR appeared successful for HDM control (Howieson *et al.,* 2002; Htut *et al.*, 1996; and McIntyre, 1992). However, not all of the studies measured ventilation rates nor the clinical efficacy of the remedial measures.

Howieson *et al.* (2002; 2003) examined the effect of a number of remedial measures (including MVHR, steam cleaning, new bedding) on 54 asthmatic subjects in North Lanarkshire. The study concluded that lung function measurements and health questionnaire data confirmed a significant improvement in the active group compared with the control group. However, the study presented a number of confounding variables. For example, no pressure tests were carried out. In addition, no skin prick tests were undertaken and consequently the project could not differentiate between the health effects influenced by a reduction in HDM allergen levels and/or the overall improvement on indoor air quality produced by greater ventilation rates.

In another UK research project adopting MVHR, twenty houses in the Southampton area were fitted with MVHR and a further 20 houses acted as controls (Stephen *et al.,* 1997; Warner *et al*, 2000). As air-infiltration was also measured, the effect of air-leakage on indoor humidity was explored using linear regression. It emerged that the houses group with MVHR showed a significant effect of leakiness with leaky houses having lower humidity. In the control houses, however, it appears that humidity was not affected by the houses' leakiness. However, the independent analysis of the two groups does not show that the two results are statistically different from each other. As regards the effect of MVHR on allergen concentration, Warner *et al* (2000) noted that there was evidence for a beneficial effect of MVHR on *Der p1* levels. However, the reduction in allergen levels did not result in a significant clinical improvement - the power of this study was low to detect clinical changes. "The likelihood of being able to show a change in clinical symptoms could be improved by performing a larger study and ventilating more areas of the houses, possibly with the inclusion

of active dehumidification within the systems." (*ibid*).

Many studies do not attempt to investigate the links between ventilation and health but rather between *dampness* and health. Ventilation and damp can be closely related and so such studies are reported here. There is however, a lack of clarity in the literature as to the definition of a 'damp' building. For example, high absolute humidity, high relative humidity, high moisture content of elements of the fabric and the occurrence of mould are all possible indicators. This definition is critically important because whereas, for example, the absolute humidity will always tend to drop with increased ventilation (unless the external absolute humidity is higher), relative humidity is a function of both temperature and the moisture content of the air and so increased ventilation can, under some circumstances, increase relative humidity.

A recent report (ISBE, 2003) concluded that extensive knowledge exists on the influence of humid environments to human health. Health problems in damp and humid buildings were first described by Leeuwen (1924) and these problems related to buildings have received increased attention over the last few decades. In the last two decades several large studies in the UK, USA, Scandinavian Countries and the Netherlands strengthened confidence in the relationship between indoor environmental humid conditions and an increase in asthma, impaired respiratory function, general respiratory symptoms and respiratory infection among children (Brunekreef *et al* 1993; Dales *et al* 1991; Cuijpers *et al* 1995; Li and Hsu 1996 and Rylander 2003)).

A recent review of the literature on dampness and HDM exposure in buildings and health effects (Bornehag *et al* 2004) concluded that dampness is a risk factor for health in domestic environments but that the literature is not conclusive in respect of causative agents. The strong need for more multidisciplinary studies was noted.

A recent study (Hagerhed *et. al.,* 2002) reported that dampness (inferred from visible signs of mould and condensation, coupled with the perception of indoor air quality e.g. 'stuffy air') is more common in older buildings and buildings with natural ventilation. Bornehag (2002) summarised 15 different studies on dampness and health concluding that in 13 studies a positive association was found between dampness and health effects namely asthma and wheezing.

A study conducted in Scotland (Williamson, 1997) showed that asthmatic patients attending a hospital asthma clinic were two to three times more likely to live in a dwelling with evidence of dampness (inferred from fabric moisture content and severity of mould) than an age and sex matched random sample of the general population living in the same area of the city of Glasgow.

Further confirmation of the significant influence of house dampness and mould on health status is reported in numerous, mainly medical, reports and journals (Garrett *et. al.*, 1998; Andriessen, *et. al.*, 1998; Zock, *et. al.*, 2002; Zureik, *et. al.*, 2002).

As noted earlier, some of the studies reported here do not deal directly with ventilation – rather 'damp' housing. The two are related but it is clear that the number of studies which have attempted to link ventilation rates directly to respiratory problems are scarce.

It may be helpful to conclude this section with the findings of the National Academy of Sciences, USA. The results of workshops in 1999 (Committee, 2000) reported 'existing data are inadequate for conclusions regarding the association between ventilation rates or ventilation system microbiological contamination and either the exacerbation of asthma symptoms or asthma development.' 'Airtight building envelopes and low rates of ventilation have been cited as factors that may contribute to asthma incidence or symptoms or may explain recent increases in asthma; however, very few relevant data are available ... .measurements of ventilation rates should be included, when possible, in future asthma case-control studies or cross-sectional surveys.'

5. Discussion

The literature review highlights the limited research that has been undertaken to demonstrate a *direct* relationship between domestic ventilation and health. The medical and building science literatures include many publications addressing ventilation and health in

offices, but few domestic housing studies. There have been studies by occupational health services to support the growing white-collar workforce but because private housing is not regulated by health and safety legislation there are fewer relevant studies. Also, it is relatively difficult to measure ventilation in housing: current techniques can only be undertaken on a small number of dwellings at a time. Yet, health studies generally require large occupant samples.

Health effects of pollutants can be studied in three ways – medicine, toxicology and epidemiology. Typically, the first is of concern in relation to an individual person. The person has unexpected symptoms, and explanations for these are sought in the surrounding environment. If a particular aspect of the environment is suspected, then two further directions are possible: toxicological studies will test the possible agent in laboratory settings (for example, effects on tissue cultures or mice); epidemiological studies will compare the frequency of the disease in people exposed to the pollutant with people not exposed.

This approach has served industrial medicine well, and has identified potential hazards of particular working environments, for example dyes in the chemical industry causing bladder cancer and asbestos in the construction industry causing lung mesothelioma. But assessing the effects of pollutants in the home is more complicated. People living at home have a varied environmental exposure (living in different rooms, working with different materials, periods of time in and out of the house) and these are not usually recorded before the onset of symptoms. The levels of exposure are probably below industrial levels, and health records not so accessible. In general, whereas occupational health services assess the health of workers in the workplace, general medical services rarely see people in their homes except for nursing and care services.

In the industrial examples given above, the diseases identified were relatively unusual. On the other hand, respiratory symptoms that may be attributed to housing are widely prevalent. Coughs and colds affect most people every year. Moderate or severe symptoms can arise from infections (bronchitis and pneumonia) and 'asthma', (a state of narrowing of respiratory airways). The United Kingdom has the largest proportion of people in Europe who believe they have asthma – one in three of the population by current surveys. (Whether the country comparisons are accurate is another question: the rapid growth in self-diagnosis of asthma suggests a possible re-allocation of other respiratory complaints {coughs and colds}). Respiratory symptoms are common; and so also are the various factors suggested to cause them. To focus on asthma again, the range of possible 'causes' include respiratory infections, chemical vapours in the air, ingested chemicals, dust-borne particles, temperature changes, smoking, exercise and psychological reactions. Asthma symptoms occur because of inflammation of the respiratory tract. 'Allergy' is only one of several pathways for the body to create the inflammation, and people who are 'allergic' don't necessarily have that as the cause of specific asthma symptoms – which may be the result of a simple viral infection (cold, cough) or increased obesity making exercise more difficult.

Good studies relating home air to respiratory symptoms are difficult to achieve. Symptoms are common and difficult to standardise, while exposures are equally hard to record. Typically, studies have tried to focus on asthma as a disease, since it occurs in children and there appear to be a wide variety of triggers. Three sorts of epidemiological studies can be used: Cross-sectional studies are most common. These surveys record existing exposures in housing and existing levels of disease, and look for statistical associations between the two. The weakness in these studies is 'confounding' – that is, several factors may be identified statistically, but another factor (perhaps unrecorded) may be having a greater real effect. Other criteria, including strength of association, biological plausibility and specificity of the effect should be considered in evaluating the results.

Case-control studies compare people with symptoms against people without symptoms, and look for different levels of exposure. These studies can look at past exposure, and therefore show whether exposure happened before disease – which cross-sectional studies cannot. Nevertheless, it is necessary to collect information on all possible causes, and still the case-control design does not exclude confounding factors.

Longitudinal population studies have the strongest scientific design. They follow a cohort of people forwards in time. They measure the exposure before onset of the disease, and therefore help avoid confounding factors. However, they have to be large, since only a proportion of people tracked over time will develop the symptoms. This in turn means measuring ventilation over a large number of properties which is very expensive.
In the field of domestic ventilation and health so far, only two sides of the equation have been assessed in detail: there have been studies of the effects of ventilation on air characteristics, such as dust mites, mould spores, carbon monoxide; and there have been studies showing how these factors are associated with respiratory symptoms and diseases. But, in contrast to office studies, there have been relatively few *direct* studies of domestic ventilation and diseases.

It may be reasonable to accept the separation of the 'technical' studies of ventilation from the 'medical' studies of air and disease. Thus, if ventilation reduces pollutants, then potential harm may be reduced. But it would be welcome to know whether there was a real effect because the implications of ventilation are not negligible. Energy efficiency seeks to maintain indoor temperatures while reducing heat loss. 'Tight' buildings have reduced levels of natural ventilation; but they may have increased pollutants, for example, high concentrations of domestic chemicals (e.g. cleaning materials), cooking gases (carbon monoxide) or house dust mites (through higher humidity). These in turn are believed to have respiratory symptom consequences, but the link to levels of ventilation is unknown – making it difficult to give appropriate guidance.

Two other points are relevant for the discussion. First, the concept of 'attributable risk'; this suggests how much a factor contributes to a disease (attributable fraction) or how many people may be affected (population attributable fraction). While the attributable fraction may be low – say only one in twenty cases, if the disease is widespread then the population effect can be quite large in number of cases. (Take, for example, the view that current levels of external air pollution are causing more than 10,000 premature deaths in Britain each year.) A scientific study of ventilation would wish to make some numerical estimate of the health effects.

Second, it is probable that there is a threshold level for some pollutants. Equally, it is possible for human bodies to excrete or detoxify some chemicals. We do not have adequate knowledge of thresholds and should not assume that only a perfectly 'clean' environment is healthy. Ventilation itself is not a health hazard. However there is considerable theoretical evidence to support the hypothesis that ventilation rate around the levels controlled by the Building Regulations can have a health effect. The pathways are summarised in Figure 1. However, this diagram hides the complexity in trying to identify if a real link exists. Ventilation may impact on hazards and then respiratory problems which are not only affected by spores and faeces but also by other hazards. This is made more complex by the real difficulties in monitoring ventilation, hazards and respiratory problems in one study. It is these difficulties in monitoring that may, in part, explain the often conflicting results from different studies. Being able to monitor ventilation, HDM populations, mould growth and respiratory problems is key to determining a link. The difficulties in monitoring respiratory problems are discussed above but an important issue also relates to most studies relying on self reporting rather than any medical diagnosis.

Measuring the actual hazards (mould and house dust mites) is also problematic. Although the English House Condition Survey has developed a method to rate the severity of mould growth by surveyors, many studies simply rely on the occurrence of any mould, condensation or damp - terms which can have a wide range of different interpretations. It is possible for example to hypothesise a scenario where the occurrence of condensation could *reduce* mould and house dust mites. For example, condensation on single glazing may be a useful stimulus for occupants to increase occupant controlled ventilation which in turn would lead to less mould and HDM. The advantage of monitoring mould is that it is at least visible and so self reporting is possible. However, there is the complexity that mould may be evident due to a previous problem which no longer exists if it has not been cleaned up. In the case of HDM, these are not visible and monitoring is very difficult. HDM populations also vary seasonally due to the change in outside vapour pressure. Sampling is very dependent on the methods used, e.g. vacuum cleaning area of the building,

since mites are invisible there may be a colony of mites which happen to have the correct micro-environment in a location which has not been sampled. Also it is not the mites themselves that cause the respiratory problems but their faeces which have a long life and can be present even if the colony has been eradicated.

As noted earlier, domestic ventilation is also very difficult to measure on the scales required and no reliable methods of inferring domestic ventilation rates have been developed. The most commonly measured parameter to infer ventilation rates is pressure testing. However, pressure testing only reveals something about the background air infiltration and nothing about the occupant controlled ventilation.

6. Conclusions

A key change in this field in recent years has been a move from extrapolating from laboratory studies of air pollution causing respiratory symptoms to the use of a limited number of epidemiological studies of the real world for long-term effects.

An extensive body of literature exists which attempts to investigate relationships between ventilation and indoor air quality. There is general consensus that a link exists between ventilation rates in dwellings and respiratory hazards (for example HDM). There is also general consensus that a link exists between these respiratory hazards and respiratory problems. For relevant moisture related respiratory hazards (HDM and fungal growth), the literature offers some advice on the minimum required ventilation rates to prevent unacceptable hazard and the consequent respiratory health risks.

Of particular interest though, it appears that most existing data are inadequate for conclusions to be drawn regarding the *direct* association between ventilation rates and respiratory problems. It is noted that there are many real difficulties in attempting to establish such a relationship and further work may be required to achieve this

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8. References

Adan, O., Schober, G., Kniest, F. and Vorenkamp, J. (1988) "Modification of indoor humidity conditions: an allergological sanitation method for the indoor environment", *Revue Francaise D'Allergologie et D'Immunologie Clinique*, Vol. 28, pp. 147-151.

Adeeb, F., Shooter, D.(2003) "Emission and evolution of air-borne microflora in slaughter houses", *Indoor-Built Environ,* Vol. 12, pp. 179-184.

Andriessen, J.W., Brunekreef, B., Roemer, W. (1998) "Home dampness and respiratory health status in European children", *Clinical and Experimental Allergy*, Vol. 28, pp. 1191-1200.

Arlian, L.G., Platts-Mills, T.A. (2001) "The biology of dust mites and the remediation of mite allergens in allergic disease", *Journal of Allergy Clinical Immunology*, Vol. 107 (3 Suppl.), pp. S406-13.

Berry, R.W., Brown, V.M., Coward, S.K.D., Crump, D.R., Gavin, M., Grimes, C.P., Higham, D.F., Hull, A.V., Hunter, C.A., Jeffery, I.G., Lea, R.G., Llewellyn, J.W. and Raw, G.J. (1996) "Indoor air quality in homes: part 1. The Building Research Establishment Indoor Environment Study", London, Construction Research Communications.

Bornehag, CG, Sundell, J, Bonini, S, Custovic A, Malmberg P, Matricardi, P, Skerfving

S, Sigsgaard, T & Verhoeff, A. (2004) Dampness in buildings as a risk factor for health effects, EUROEXPO. A multidisciplinary review of the literature (1998–2000) on dampness and mite exposure in buildings and health effects Indoor Air Vol. 14(4) pp. 243 - 257.

Bornehag, C.G. and Sundell, J. (2002) "Dampness in buildings as a risk factor for health effects", in: *Proceedings: Indoor Air 2002. 9th International Conference on Indoor Air Quality and Climate*, Monterey, Indoor Air 2002.

Brunekreef, B., Verhoeff, R.T., van Strien and van Wijnen. (1993) "The role of sensitisation to dust mites and moulds in explaining the relationship between home dampness and childhood respiratory symptoms", in: *Proceedings of Indoor Air 93: 6th international conference on indoor air and climate*, Vol 1, pp 153-158.

Building Research Establishment, (1990), "Research project number EP228", Garston, BRE.

Call, R.S., Smith, F.T., Morris, E. Chapman, M.D., Plat-Mills, T.A.E. (1992) "Risk factor for asthma in the inner city children", *The journal of paediatrics*, Vol 121, pp. 862-866.

Chapman, J.A., Terr, A.I., Jacobsn R.L., Charlesworth, E.N., Bandana, E.J. (2003) "Toxic mould: phantom risk vs. science", *Ann Allergy, Asthma and Immuno.*, Vol. 91, pp. 222-232.

Committee on the Assessment of Asthma and Indoor Air. (2000) "Clearing the Air", Washington, National Academy Press.

Comtois, P. and Garcia, B.E. (1994) "Indoor mycology the Northern American Experience", in: Singh, J (Edited by), *Building Mycology, Management of Health and Decay in buildings*, London, E & FN Spon, pp. 260-280.

Crowther, D., Horwood, J., Baker, N., Thomson, D., Pretlove, S., Ridley, I., Oreszczyn, T., (2000), "House Dust Mites and the Built Environment: a Literature Review", available online at: www.arct.cam.ac.uk/research/mite/review.pdf.

Crowther, D., Pretlove, S., Ridley, I., Oreszczyn, T., Wilkinson, T. (2002) "A Hygrothermal Model of House Dust Mite Response to Environmental Conditions in Dwellings Summary Report", available online at: http://www.arct.cam.ac.uk/research/mite/four.pdf.

Cuijpers, C.E.J., Swaen, G.M.H., Wessling, G., Sturmans, F., Wouters, E.F.M. (1995) "Adverse effects of the indoor environment on respiratory health in primary school children", *Environ Res*, Vol. 68, pp. 11-23.

Cunningham, M. J. (1996) "Controlling Dust Mites Psychrometrically - a Review for Building Scientists and Engineers", *Indoor Air*, Vol. 6, pp. 249-258.

Dales, R., Zwanenburg, H. and Burnett, R. (1990) "The Canadian air quality health survey: Influence of home dampness and moulds on respiratory health", in: *Proceeding of Indoor air '90. 5th international conference on Indoor air quality and climate,* Ottawa, Indoor Air 90, Vol. 1, pp 145-147.

Department of Environment, Transport and Regions, (1996) "English House Condition Survey 1996", London, DETR.

Emenius G., Svartengren M., Korsgaard J., Nordvall L., Pershagen G. and Wickman M. (2004) Building characteristics, indoor air quality and recurrent wheezing in very young children (BAMSE), Idoor Air Vol. 14(1) pp. 34 - 42.

Emenius, G., Egmar, A., Wickan, M. (1998) "Mechanical ventilation protects one-storey single-dwelling houses against increased air humidity, domestic mite allergens and indoor pollutants in a cold climatic region", Clinical *and Experimental Allergy*, Vol. 28(11), pp. 1389-96.

Flannigan, B., MaCabee, E.M., McGarry, F. and Strachan, D.P. (1990) "Wheeze in children: An investigation of air spora in the home", in: *Indoor air '90. 5th international conference on Indoor air quality and climate,* Ottawa, Indoor Air 90, Vol. 2 pp. 27-32.

Fletcher, A.M., Pickering, C.A., Custovic, A., Simpson, J., Kennaugh, J., Woodcock, A. (1996), "Reduction in humidity as a method of controlling mites and mite allergens: the use of mechanical ventilation in British domestic dwellings", *Clinical and Experimental Allergy*, Vol. 26(9), pp.1051-6.

Garrett, M.H., Rayment, P.R., Hooper, M.A., Abramson, M.J., Hooper, B.M. (1998) "Indoor airborne fungal spores, house dampness and associations with environmental factors and respiratory health in children", *Clinical and Experimental Allergy*, Vol. 28, pp. 459-467.

Gotzsche, P.C., Hammrquist, C., Burr, M. (1998) "House dust mite control measures in the management of asthma: meta-analysis", *British Medical Journal*, Vol. 317, pp.1105-1110.

Hagerhed, L., Bornehag, C.G., Sundell J. (2002) "Dampness in buildings and health. Building characteristics as predictors for dampness in 8681 Swedish dwellings", in: *Proceedings: Indoor Air 2002. 9th International Conference*

on Indoor Air Quality and Climate, Monterey, Indoor Air 2002.

Hart, B.J. and Whitehead, L. (1990) "Ecology of house dust mite in Oxfordshire", *Clinical and Experimental Allergy*, Vol. 20, pp. 203-209.

Harving, H., Korsgaard, J., Dahl, R. (1994) "Clinical efficacy of reduction in house-dust mite exposure in specially designed, mechanically ventilated "healthy" homes", *Allergy*, Vol. 49(10), pp.866-70.

Howieson, S.G., Lawson, A., McSharry, C., Morris, G., McKenzie, E. (2002) "1B2O5 Indoor Air Quality, Dust Mite Allergens And Asthma", in: *Proceedings Indoor Air 2002. 9th International Conference on Indoor Air Quality and Climate*, Monterey, Indoor Air 2002, pp.113-118.

Howieson, S.G., Lawson, A., McSharry, C., Morris, G., McKenzie, E., Jackson, J. (2003) "Domestic ventilation rates, indoor humidity and dust mite allergens - Are our homes causing the asthma pandemic", *Building Services Engineering, Research and Technology Journal*, Vol. 24, No. 3, pp.137-147.

Htut, T., Burgess, I.F., Maunder, J.W., Basham, E. (1996) "A pilot study on the effect of one room mechanical ventilation with heat recovery (MVHR) units on house dust mite populations and Der p 1 levels in laboratory simulated bedrooms and on ambient conditions in an occupied bedroom in Cambridge, UK", *International Journal of Environmental Health Research*, Vol. 6, pp.301-313.

Hunter, C.A., Grant C., Flannigan B. and Bravery A.F. (1988) "Mould in buildings: the air spora of domestic buildings", *International Biodeterioration,* Vol. 24, pp. 81-101.

Hunter, C.A., Hull A.V., Higham, D.F., Grimes, C.P. and Lea, R.G. (1996) "Fungi and bacteria" in: Berry, R. W., Brown, V. M., Coward, S. K.D., Crump, D. R., Gavin, M., Grimes, C. P., Higham, D. F., Hull, A. V., Hunter, C. A., Jeffery, I. G., Lea, R. G., Llewellyn, J. W. and Raw, G. J., (1996), *BRE Report 299. Indoor air quality in homes: part 1*, Garston, BRE; pp 97-115.

Husman, T., Koskinen O., Hyvarinen A., Reponen T., Ruuskanen J. and Nevalainen A. (1993) "Respiratory symptoms and infections among residents in dwellings with moisture problem or mould growth", in: *Proceedings of indoor air 93; 6th international conference on indoor air quality and climate,* Vol 1, pp171-174.

Irie, T., Hasegawa, Y. and Yoshikawa, M. (1990) "Measurement of living mites in private wooden dwellings, Japan", in: *Proceedings International Conference on Indoor Air Quality and Climate*, Ottawa, Vol. 2, pp 61-66.

ISBE (International Society of the Built Environment), (2003) "Workgroup Report", *Indoor and Built Environment,* Vol. 12(4), pp. 259-264.

Kalliokoski, P. (2003) "Risks caused by airborne microbes in hospitals – source control is important", *Indoor-Built Environ.*; Vol. 12, pp. 41-46.

Kemp, P.C., Neumeister-Kemp H.G., Kock, C., Lysek, G., Murray, F. (2002a) "Determining the Growth and Vitality of Micro-Organisms in Carpets and Mattresses in Non-Problem Dwellings by Measuring CO2 Released during Respiration", *Indoor-Built Environ*, Vol.11, pp.214-220.

Kemp, P.C., Neumeister-Kemp H.G., Murray, F., Lysek, G. (2002b) "Airborne Fungi in Non-Problem Buildings in a Southern-Hemisphere Mediterranean Climate: Preliminary Study of Natural and Mechanical Ventilation", *Indoor-Built Environ.,* Vol. 11, pp. 44-53.

Korsgaard, J. (1979) "The effect of indoor environment on house dust mite", in: P O Fanger and O Valbjorn, (Edited by), *Indoor Climate*, Copenhagen, Danish Building Research Institute, pp 187-199.

Lacey, J. (1994) "Indoor aerobiology and health", in: Singh, J., (Edited by), *Building Mycology, Management of Health and Decay in buildings,* London, E & FN Spon, pp. 77-130.

Lange, J.H., Grad, J.W., Lange, P.A., Thomulka, K.W., Dunmyre, G., Lee, R.J., Richardson, C.F., and Blumershire, R.V.H. (1993) "Asbestos abatement of ceiling panels and mould growth in a public school building after water damage: a case study", *Fresenius Environ Bulletin,* Vol. 2, pp. 13-18.

Leeuwen, W.S. (1924) "Bronchial asthma in relation to climate", *Proc Roy Soc Med*, pp. 17-19.

Li, C.S. and Hsu, L.Y. (1996) "Home dampness and childhood respiratory symptoms in a subtropical climate", *Arch Environ Health*, Vol. 51, pp. 42-46.

Lowe, R. (2000) "Psychrometric control of Dust mites in UK Housing", *Building Services Engineering Research and Technology*, Vol. 21, No. 4, pp.274-276.

Lugauskas, A., Krikstapois, A., Seskauskas, V. (2003) "Species of conditionally pathogenic Micromycetes in the air of dwelling and occupational premises", *Indoor-Built Environ*, Vol.12, pp. 167-178.

McIntyre, D.A. (1992) "The control of house dust mites by ventilation: a pilot study", *Proc. 13th AIVC Conf.*, Nice, AIVC, pp.497-507.

Menetrez, M.Y., Forde K.K., DeJesus V.R. (2002) "Testing Antimicrobial Efficacy on Porous Materials", *Indoor-Built Environ.*, Vol.11, pp. 202-207.

Menetrez, M.Y., Foarde, K.K. (2002) "Microbial Volatile Organic Compound Emission Rates and Exposure Model", *Indoor-Built Environ.*, Vol. 11, pp. 208-213.

Minnesota Department of Health (2001) "Recommended best practices for mould investigations in Minnesota schools", Environmental Health Division, Indoor Air Unit, St. Paul, Mn 55164.

Mussalo-Rauhamaa, H., Suomalainen, H., Helin, T., Elg, P., Orpana, A. (2003) "Bronchoalveolar lavage findings from persons exposed to moulds in water-damaged houses", *Indoor-Built Environment*, Vol. 12, pp. 237-237.

New York City Department of Health (2000) "Guidelines on assessment and remediation of fungi in indoor environments", New York, Bureau of Environmental and Occupational Disease Epidemiology.

Niven, R., Fletcher, A.M., Pickering, A.C., Custovic, A., Sivour, J.B., Preece, A.R., Oldham, L.A., Francis, H.C. (1999) "Attempting to control mite allergens with mechanical ventilation and dehumidification in British houses", *Journal of Allergy Clinical Immunology*, Vol. 103 (5 Pt 1), pp. 756-62.

Office of the Deputy Prime Minister (ODPM), (2003), "English House Condition Survey 2001", ODPM, London.

Peat, J.K., Dickerson, J., Li, J. (1998) "Effects of damp and mould in the home on respiratory health: a review of the literature", *Allergy*, Vol. 53, pp. 120-128.

Platts-Mills, T.A.E. and De Weck, A.L. (1989) "Dust mite allergens and asthma: A world wide problem", *J Allergy Clin Immunol.*, Vol. 83, pp. 416-27.

Pope, A.M., Patterson, R. and Burge, H. (Edited by) (1993) "Indoor allergens: assessing and controlling adverse health effects", Washington, National Academy Press.

Rao, C.Y., Burge, H.A., Chang, J. (1996) "Review of quantitative standards and guidelines for fungi in indoor air", *J Waste Manag Assoc.*, Vol. 46, pp. 899-908.

Rautuiala, S., Reponen, T., Nevalainen, A., Husman, T., Kalliokoski, P. (1998) "Control of exposure to airborne viable microorganisms during remediation of mouldy buildings: report of three case studies", *Am J Ind Hyg.*, Vol. 59, pp. 455-460.

Raw, G.H. and Fox, T.A. (1990) "Condensation heating and ventilation in small homes", in: *CIB Conference. Energy moisture and climate*, Rotterdam, CIB.

Raw, G.J., Aizlewood, C.E. and Hamilton, M. R. (2001) "Building Regulation, Health and Safety", Garston, BRE.

Ridley, I., Fox, J., Oreszczyn, T., Houng, S.H. (2003) "The impact of replacement windows on air infiltration and indoor air quality in dwellings", *The International Journal of Ventilation*, Vol. 1, No. 3, pp. 209-218.

Rylander, R. (2003) "Humid building – the problem", *Indoor and Built Environment.*, Vol. 12, pp. 211-213.

Sarca, S, Asan, A, Otkun, MT, Ture, M. (2002) "Monitoring Indoor Airborne Fungi and Bacteria in the Different Areas of Trakya University Hospital, Edirne, Turkey", *Indoor-Built Environ.*, Vol. 11, pp. 285-292.

Singh, J. (1994a) "Building Mycology Management of Health and Decay in buildings", London, E & FN Spon.

Singh, J. (1994b) "Allergy Problem in Buildings", Lancaster, Quay Publishing.

No.1, pp. 22-34.

Singh, J. (1999) Sick Building Syndrome, Facilities Management, Health and Safety, November issue, pp. 12-15.

Spengler, J., Neas, L., Nakai, S., Dockery, D., Speizer, F., Ware, J., and Raizenne, M. (1993)

"Respiratory symptoms and housing characteristics", in: *Proceedings of indoor air 93; 6th international conference on indoor air quality and climate*, Vol 1, pp. 165-170.

Stephen, F. R., McIntyre, D. A., Lane, A., Raw, G.J., Wiech, C.R., Frederick, J. (1997) "Ventilation and House Air Tightness: Effect on Indoor Temperature and Humidity in Southampton, UK", *BSERT* (Building Services Engineering Research Technology Journal), Vol.18, No.3, pp.141-147.

Strachan, D.P (1998) "House dust mite allergen avoidance in asthma. Benefits unproved but not yet excluded", *British Medical Journal*, Vol. 317, pp.1096-1097.

Su, H.L., Spengler, J.D., and Burge, H.A. (1990) "Examination of microbiological concentration and examination with childhood respiratory health", in: *Proceedings of indoor air 90; 5th Indoor international conference on indoor air and climate,* Ottawa, Indoor Air 90, Vol. 2, pp. 21-26.

Sundell, J., Wickman, M., Pershagen, G., Nordvall, S.L. (1995) "Ventilation in homes infested by house-dust mites", *Allergy*, Vol. 50, No. 2, pp.106-12.

Toma, T., Miyagi, I., Kishimoto, M., Nagama, T., and Tamanaha, I. (1993) "Fauna and seasonal appearances of mites in house dusts collected from the residences including asthmatic children in Okinawa Prefecture, Ryukyu Archipelago", *Japanese Journal of Sanitary Zoology*, Vol. 44, pp. 223-225.

Wargocki, P., Sundell, J., Bischof, W. (2002) "Ventilation and health in non-industrial indoor environments: report from a European Multidisciplinary Scientific Consensus", *Indoor Air*; Vol. 12, No. 2, pp. 113-8.

Warner, J., Ferderick, J., Bryant, T., Weich, C., Raw, G., Hunter, C., Stephen, F., McIntyre, D. and Warner, J. (2000) Mechanical ventilation and high efficiency vacuum cleaning: a combined strategy of mite and mite allergen reduction in the control of mite-sensitive asthma, J.Allergy Clin. Immunol., 106(No. 1, Pt 1), 75-82.

White, J.H. (1990) "Solving moisture and mould problems", in: *Proceedings of indoor air 90; 5th international conference on indoor air quality and climate,* Ottawa, Indoor Air 90, Vol. 4, pp. 589-594.

Williamson, I.J., Martin, C.J., McGill, G., Monie, R.D.H., Fennerty, A.G. (1997) "Damp housing and asthma: a case control study", *Thorax*, Vol. 52, pp. 229-234.

Woolliscroft, M., (1997), "PH-97-8-3. Residential Ventilation in the United Kingdom: An Overview", *ASHRAE Transactions: Symposia,* Vol. 103, No. 1, Paper Number: PH-97-8-3, pp. 706-716.

Zock, J.P., Jarvis, D., Luczynska, C., Sunyer, J., Burney, P. (2002) "Housing characteristics, reported mould exposure, and asthma in the EC respiratory health survey", *Journal of Allergy and Clinical Immunology*, Vol. 110(2) pp. 285-292.

Zureik, M., Neukirch, C., Leynaeret, B., Liard, R., Bousquet, J., Neukirch, F. (2002) "Sensitisation to airborne moulds and severity of asthma: cross sectional study from EC respiratory health survey", *BMJ*, Vol. 325 (7361) pp. 411-414.

Appendix A.0: Published Papers

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A.0.2: *Predicting the population dynamics of the house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle*

Phillip Biddulph[1,5], David Crowther[1], Brian Leung[2], Toby Wilkinson[1,3], Barbara Hart[4], Tadj Oreszczyn[5], Stephen Pretlove[6], Ian Ridley[5], **Marcella Ucci**[5]

[1]The Martin Centre, University of Cambridge, 1 Scroope Terrace, Cambridge CB2 1PX, UK
[2]Biology Department, McGill University, 1205 Docteur Penfield, Montreal, Quebec H3A 1B1, Canada
[3]Medical Entomology Centre, Barrington Road, Shepreth, Royston SG8 6QZ, UK
[4]Royal Agricultural College, Stroud Road, Cirencester, Gloucestershire, GL7 6JS,UK
[5]The Bartlett School of Graduate Studies, University College London, WC1E 6BT, UK
[6]Kingston University, School of Architecture & Landscape, London, KT1 2QJ, UK

Abstract
A generalised model of the life cycle of a house dust mite, which can be tailored to any particular species of domestic mite, is presented. The model takes into account the effects of hygrothermal conditions on each life cycle phase. It is used in a computer simulation program, called POPMITE, which, by incorporating a population age structure, is able to predict population dynamics.

The POPMITE simulation is adapted to the *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) (DP) mite using published data on the egg development period, total development period, adult longevity, mortality during egg development, mortality during juvenile development, and fecundity of individual DP mites held at a range of constant hygrothermal conditions.

An example is given which illustrates how the model functions under constant hygrothermal conditions.

A preliminary validation of POPMITE is made by a comparison of the POPMITE predictions with published measurements of population growth of DP mites held at a range constant hygrothermal conditions for 21 days.

The POPMITE simulation is used to provide predictions of population growth or decline for a wide range of constant relative humidity and temperature combinations for 30 and 60 days.

The adaptation of the model to correctly take account of fluctuating hygrothermal conditions is discussed.

Keywords

House dust mites, population model, relative humidity, temperature, life cycle, population structure

Introduction

The house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) (DP) is one of several species giving rise to allergens that play a major role in allergic disease, especially asthma (Voorhorst et al. 1969 and Ford et al. 1985). DP is the most common species of house dust mite in the UK, and is predominant in many countries around the world (Bronswijk 1981 and Colloff 1998). Their major habitats are beds, carpets and soft furnishings.

Mites have few natural predators, so that in a typical habitat the principal limits on the population size are the available food and the hygrothermal environment, which can be described by the relative humidity (RH) and the temperature (T) (Bronswijk 1981). In this paper we make the simplifying assumption that food is plentiful and is not limiting the population size.

Predicting the population dynamics in the hygrothermal conditions of the habitat is a crucial step in the study of different strategies for controlling mite populations, and therefore the production of allergen (see Crowther et al. 2006). Past, present and simulated future climate data can be used in conjunction with building simulations (for example see EnergyPlus 2004) to predict the internal climate of bedrooms for a wide range of house types and conditions, as well as different occupant behaviour regimes. These predictions or actual measurements can be used in turn to predict the internal conditions of beds (Pretlove et al. 2005).

An accurate computer simulation of the population dynamics of dust mites is needed to complement the range of simulations available to predict the environment of the habitat. The suite of simulations can then be used to test scenarios for controlling dust mite populations in beds. Small changes in occupant behaviour or building design may have a significant effect on the population dynamics.

Previous attempts to model the population dynamics by Cunningham (2000) and more recently Crowther et al. (2006) have used the simplifying assumption that there exists a population growth multiplication factor which is constant for a particular RH and T combination, regardless of the structure of the population. Whilst these models have the advantage of being easy to implement they can lead to misleading results even during periods of constant conditions. For example a population of pure eggs is unable to grow until at least one mating pair has matured and as a population develops the numbers of egg laying females will fluctuate naturally. This would lead to fluctuating rates of population growth, which would cause problems for these models, both in terms of calibration and use. To overcome them a model which can keep track of the population structure has been developed.

Mites develop through various life phases, from eggs to juveniles (larvae, protonymphs and tritonymphs for DP) to adults. Mites at each phase of the life cycle have different development times and mortalities depending on the hygrothermal

conditions (Gamal Eddin et al. 1983a,c). The fecundity of female adults also depends on the hygrothermal conditions (Gamal Eddin et al. 1983b).

The population dynamics of dust mites is most sensitive to the parameters described above. As with any model, this model uses simplifying assumptions, particularly for the less sensitive parameters (e.g. the age dependence of the egg laying rate). A preliminary validation of the model indicates that these assumptions are acceptable.

A generalised model of the life cycle of a house dust mite, using information on development times and mortalities, can be tailored to any particular species of domestic mite. The age structure of the mite population can be simulated by keeping track of the development and numbers of mites in batches as they progress through the life cycle model.

In this way a computer simulation program able to predict population dynamics, called POPMITE, has been constructed that incorporates the life cycle model and a population age structure. POPMITE has been tailored to DP mites using a selection of published data describing their physiological response to a range of constant hygrothermal conditions.

Nomenclature

The following table lists the symbols, their meanings and units as used throughout this paper.

Symbol	Meaning	Units
T	Temperature	°C
RH	Relative humidity	%
t	Time slice	hour
N_{egg}	Number of eggs	eggs
$N_{juvenile}$	Number of juveniles	juveniles
N_{adult}	Number of adults	adults
D_{egg}	Egg development duration	ours
$D_{juvenile}$	Juvenile development duration	hours
D_{adult}	Adult longevity	hours
R_{egg}	Egg Percentage Development rate	%/hour
$R_{juvenile}$	Juvenile Development rate	%/hour
R_{adult}	Adult Ageing rate	%/hour
d_{egg}	Percentage development of eggs	%
$d_{juvenile}$	Percentage development of juveniles	%
d_{adult}	Percentage development of adults	%
M_{egg}	Total mortality during the egg phase.	%
$M_{juvenile}$	Total mortality during the juvenile phases.	%
M_{adult}	Total mortality during the adult phase.	%
s_{egg}	Egg survivability, the probability of surviving until hatching.	
$s_{juvenile}$	Juvenile survivability, the probability of a freshly hatched juvenile surviving until moulting into an adult.	
s_{adult}	Adult survivability, the probability of surviving the natural adult life span.	
S_{egg}	Egg hourly survival probability	$hour^{-1}$
$S_{juvenile}$	Juvenile hourly survival probability	$hour^{-1}$
S_{adult}	Adult hourly survival probability	$hour^{-1}$
F	Egg laying rate of adult female mites.	eggs/hour
f	Fecundity or the mean number of eggs produced per female mite during her adult life.	eggs

Theory

The methods for calculating the population dynamics described here are similar to those that use the Leslie matrix model (Leslie 1945) as described by Nisbet and Gurney (1982) and Case (2000).

If the initial structure of a population of mites (numbers and ages of eggs, juveniles and adults) is known, then our aim is to predict the change in the structure after a short period of constant hygrothermal conditions. This can be done by calculating:

- the increase in development and the loss of mites for each of the life cycle phases separately, and
- the number of freshly laid eggs.

By repeating this process over many time periods one can thus predict the population dynamics.

A population of mites will consist of batches of individuals at any one of the phases of their life cycle. Each batch will be at a unique development stage within the phase and will be progressing through the phase at a rate which depends on the hygrothermal environment. The environment will also play a role in determining the chances of survival and the rate at which adult females lay eggs. The physiological response of mites in a particular phase to the constant conditions can be described by:

- the percentage development rate (R),
- the survival probability (S), and
- for adult females, the egg laying rate (F).

It is assumed that each of these parameters R, S and F will depend only on the current hygrothermal conditions RH and T.

In the model we can make a list, or an array, of the number of mites in each batch in each phase, indexed by the percentage development $d\{t_n\}$, where t_n is the current time. So the number of mites in a batch in a phase is described as $N\{d\{t_n\}\}$, where $d\{t_n\} = 0\%$ at the beginning of the phase. To calculate how further developed this batch of mites are in the next time slice t_{n+1}, where $t_{n+1} = t_n + t_{unit}$ and t_{unit} is typically 1 hour, we use the percentage development per hour $R(RH,T)$. So the new percentage development of this batch of mites at time t_{n+1} is:

$$d\{t_{n+1}\} = d\{t_n\} + R(RH,T) \qquad (1)$$

During this time some of the mites in the batch will die. To calculate how many mites survive into the next time slice we use the survival probability per hour $S(RH,T)$. So the number of mites surviving into the next time slice is given by:

$$N\{d\{t_{n+1}\}\} = N\{d\{t_n\}\} \times S(RH,T) \qquad (2)$$

It should be noted that $N\{d\{t_{n+1}\}\}$ is not an integer and can become very much less than 1 if the conditions are bad for an extended period of time. This can be interpreted as

the probability of survival from eradication during this time slice. If $N\{d\{t_{n+1}\}\}$ is tiny (less than say 0.000001) then it is very unlikely that this batch will survive and it is removed from the list of batches.

The percentage development rate and survival rate are used in every phase of the life cycle and a subscript is now used to indicate which phase a particular parameter represents. For example $N_{egg}\{d_{egg}\{t_n\}\}$ is the number of eggs with percentage development $d_{egg}\{t_n\}$, at time t_n.

The adult phase is a special case, as in addition to the development rate and the survival rate, adult female mites may lay eggs. To calculate how many eggs are laid in the next time slice we use the eggs laid per hour $F(RH,T)$. So the number of fresh eggs laid in the next time slice is given by:

$$N_{egg}\{d_{egg}\{t_{n+1}\}\} = N_{female} \times F(RH,T), \qquad (3)$$

where $d_{egg}\{t_{n+1}\} = 0\%$, because they are freshly laid, and $N_{female} = N_{adult}/2$

It is assumed for the purposes of this model that half the adults are female (Hodgson (1976)) and that all adult females of all ages can produce eggs if the conditions are favourable. The sex ratio of eggs produced is assumed to be 1:1, and there are no differences between the sexes, other than that female mites can lay eggs. These simplifying assumptions can be relaxed if necessary.

This process is repeated each time slice with adult female mites replenishing the supply of eggs, as batches of mites progress through the phases, getting ever more developed, as well as ever more depleted in numbers as they die, until they reach a development of 100%. Once a batch of mites has reached a development of 100%, it is moved into the next phase with a percentage development of 0%. At the end of the final adult phase the mites die and are removed from the model completely.

Figure 1 is a schematic diagram showing the model of a simplified mite life cycle with only three phases, egg, juvenile and adult. Time proceeds down the page for three consecutive time slices. The RH and T are assumed to be constant throughout each time slice. The structure of each phase is depicted as separate histograms across the page with development as the x-axis and number of mites as the y-axis. Within the histogram, batches are represented by thick vertical bars. The height of each bar represents the number of eggs, juveniles or adults in a batch, and the position along the histogram shows its progress. After each time slice, batches are moved along the histograms until they reach the end and are then either moved into the next stage or, for adults, are removed altogether, representing the end of their lifespan. The bars shrink in size at every time slice depending on the survival rates. The female adults lay fresh eggs which form a new batch at the start of the next egg histogram as indicated by the dotted line.

The model naturally introduces a mix or structure into the population. The population of mites at any one moment will consist of eggs, juveniles and adults, all at different stages of development and age. The exact structure will depend on the history of the population and, to get a realistic prediction of the population dynamics over an extended period of time, it is important to keep track of this structure. For example a population experiencing a period of bad conditions leading to a much depleted population will respond very differently to a period of good conditions than a healthy population would.

The POPMITE simulation

To use the model in a simulation of a population of a real mite species, it is necessary to have values for the percentage development rate (R), the survival rate (S) for each phase in a mite's life cycle and the egg laying rate for adult females (F) for a wide range of RH and T combinations.

Data from a number of different sources has already been published describing the physiological response of the different phases of individual DP mites to a range of constant RH and T combinations. A selection of this data, suitably analysed, has been used to create a rough simulation. The data comes from different experimental groups, which use different experimental methodologies that may not be compatible. It is therefore difficult to extract anything more than an estimate of the physiological response to the hygrothermal conditions. Indeed in analysing the data our primary intention at this time is not to derive accurate relationships, but to provide data to illustrate the model. We therefore have not provided measures of the quality of the fits to the data. The authors of this paper are collecting a fuller and more consistent set of data to be used by the simulation which will potentially give a more precise prediction. The data and a description of an improved simulation will be published at a later date.

From the published data for the model we require the mathematical descriptions or formulae of the parameters:

- $R_{egg}(RH,T)$, $R_{juvenile}(RH,T)$, $R_{adult}(RH,T)$,
- $S_{egg}(RH,T)$, $S_{juvenile}(RH,T)$, $S_{adult}(RH,T)$, and
- $F(RH,T)$.

Egg development rate $R_{egg}(RH,T)$

No direct measurement of the development rate of eggs $R_{egg}(RH,T)$ was found in the literature. There is however quite detailed information on the development duration of eggs for a wide range of RH and T combinations from which the development rate can be calculated. Table 1 shows the development duration in days of DP eggs held at constant RH and T conditions.

For the purposes of the model we make the simplifying assumption that for any given combination of RH and T the development rate is constant throughout the egg life stage. We can then extract the percentage development per hour as $R_{egg} = 100\%/D_{egg}$,

where D_{egg} is the development duration in hours calculated from the data recorded in days in Table 1. Figure 2 shows R_{egg} plotted against temperature.

A linear function of temperature can be used to describe the egg development rate. Below about 50% RH no data was found on the development duration and above 50% RH no obvious correlation was found with RH (i.e. the development rate did not vary significantly for different RH values above 50% for fixed values of temperature).

The solid line on Fig. 2 is a fit to the egg development data and gives a rate of 0.91 % development per day per °C. In other words, for every 1°C increase in temperature the development rate increases by 0.91 of a percent per day. If we assume that we can extrapolate this function to lower temperatures, then development should stop completely below 9°C.

Equation 4 is the fit to the data and gives the development rate formula for $R_{egg}(RH,T)$ in % development per hour, to be used in the model.

$$\begin{aligned} R_{egg}(RH,T) &= 0.91\times(T-9)/24 \quad T>9°C \\ R_{egg}(RH,T) &= 0 \qquad\qquad\quad T<9°C \end{aligned} \tag{4}$$

The formula for the development duration of eggs will be useful later for the calculation of the juvenile development rate and the egg survival rate. This is given by equation 5, where $D_{egg}(RH,T)$ is in hours,

$$\begin{aligned} D_{egg}(RH,T) &= \frac{24\times100}{0.91\times(T-9)} \quad T>9°C \\ D_{egg}(RH,T) &= \infty \qquad\qquad\quad T<9°C \end{aligned} \tag{5}$$

Juvenile development rate $R_{juvenile}(RH,T)$

No direct measurement of the development rate of the juvenile phases of the DP mite was found in the literature. However some information for the total development duration from fresh eggs to adults is available and, by subtracting the egg development duration, the development duration of juveniles can therefore be inferred. Table 2 shows the total development duration for different combinations of RH and T. The data is from many sources, and is concentrated along the 75%RH row and the 25°C column. It can be seen that the lower the temperature, the longer the mites take to develop.

As an intermediate step we need to establish a development rate for the combined egg and juvenile life stages. In order to do so we make the simplifying assumption that, for any given combination of RH and T, the development rate is constant throughout the combined egg and juvenile development life stage. Then we can extract an percentage development per hour as $R_{total}=100\%/D_{total}$, where D_{total} is the

development time in hours calculated from the data recorded in days in Table 2. Figure 3 shows R_{total} plotted against T.

As with eggs, a linear function of the temperature can be used to describe the combined egg and juvenile development rate. Below about 50% RH no data was found on the development duration and above 50% RH no obvious correlation with development rate was found with RH. This may seem surprising given that water balance is important to mites in that above a threshold RH level they extract moisture from the atmosphere and without this moisture the mites eventually die (Arlian 1992). Mites can survive for a short period of time when the RH is below this threshold and can then recover once the RH goes above the threshold (de Boer et al. 1998). In conditions which oscillate above and below the threshold the mite development duration will depend on the length of exposure and absolute level of the low RH conditions. However, in constant conditions with RH below the threshold, mites never get the opportunity to recover and therefore die.

The solid line is a fit to the data and gives a rate of 0.33 % total development per day per °C. For every 1°C increase in temperature the development rate increases by 0.33 of a percent per day. If we assume that we can extrapolate this function to lower temperatures, then the development should stop completely below about 13°C.

$$\begin{aligned} R_{total}(RH,T) &= 0.33\times(T-13)/24 \quad T>13°C \\ R_{total}(RH,T) &= 0 \qquad\qquad\quad T<13°C \end{aligned} \tag{6}$$

The formula for overall development time from eggs to adults is given by,

$$\begin{aligned} D_{total}(RH,T) &= \frac{24\times100}{0.33\times(T-13)} \quad T>13°C \\ \mathrm{D}_{\mathrm{total}}(RH,T) &= \infty \qquad\qquad\quad T<13°C \end{aligned} \tag{7}$$

We can now subtract the egg development duration from the total development duration to get the juvenile development duration.

$$D_{juvenile}(RH,T) = D_{total}(RH,T) - D_{egg}(RH,T) \tag{8}$$

The juvenile development rate is therefore

$$R_{juvenile}(RH,T) = 100\%/D_{juvenile}(RH,T) \tag{9}$$

Adult ageing rate $R_{adult}(RH,T)$

The adult ageing rate is the rate at which both male and female adults progress towards the end of their natural life span. No direct measurements of this have been found. However table 3 gives the longevity in days of DP adults held at constant RH and T conditions.

The very sparse data there is, shows that adult mites have a much shorter life at low RH than at high RH. Figure 4 shows adult longevity as a function of temperature with two fits to the data.

The longevity of adult mites can therefore be modelled with the following equations with an arbitrary split at 60% RH. More data is needed to give a better representation of the real response of adult longevity to RH and T.

$$\begin{aligned} D_{adult}(RH,T) &= 24\times(61.42\text{-}T\times 1.31) \quad RH > 60\% \\ D_{adult}(RH,T) &= 24\times(16.44\text{-}T\times 0.32) \quad RH < 60\% \end{aligned} \tag{10}$$

The longevity data can be used to calculate the rate of ageing in % ageing per hour.

$$R_{adult}(RH,T) = 100\% / D_{adult}(RH,T) \tag{11}$$

Egg survival rate during development $S_{egg}(RH,T)$

No data for the survival rates of mite eggs during their development phase was found. However there is data on the mortality (M_{egg}) at the end of the development phase. This is defined as the percentage of mite eggs which die during the period of development and is given by this formula,

$$M_{egg} = \frac{N_{egg} - N_{juvenile}}{N_{egg}} \times 100\% \tag{12}$$

Table 4 shows the egg mortality, in percent, during the development period of mite eggs to juveniles.

The mortality is highest at low temperatures whatever the RH and also at low RH at high and low temperatures. There is clearly some discrepancy between the different datasets, especially at 25°C and 75% and 80% RH. For RH below an arbitrary cut-off, which by coincidence is also 60%, a simple quadratic of the temperature is used,

$$M_{egg}(RH,T) = 195.78 - 14.35T + 0.32T^2 \quad RH < 60\% \tag{13}$$

At RHs above 60% a more complicated exponential of the temperature is used with a simple linear formula for the RH,

$$M_{egg}(RH,T) = 31.20 - 0.19RH + 2754.05e^{-0.37T} \quad RH > 60\% \tag{14}$$

Figure 5, Figure 6 and Figure 7 show the fits to the data.

The mortality is equal to a hundred percent minus the survivability,

$$M_{egg}(RH,T) = 100\% - s_{egg} \tag{15}$$

The survivability is the product of all the hourly survival rates during the development duration, and is therefore given by

$$s_{egg} = S_{egg}{}^{D_{egg}(RH,T)/t_{unit}} \tag{16}$$

This leads to an hourly survival rate, which can be used in the model.

$$S_{egg}(RH,T) = \left(1 - \frac{M_{egg}(RH,T)}{100\%}\right)^{t_{unit}/D_{egg}(RH,T)} \tag{17}$$

It is important to note that the hourly survival rate depends on the development duration as well as the mortality of eggs.

Survival rate during the juvenile phases $S_{juvenile}(RH,T)$

No data for the survival rates of juvenile mites during their development phase was found. To calculate the juvenile hourly survival rate, the total mortality during the overall mite development from egg to adult is used. It is defined, in a similar way to the mortality during the egg development period, as the percentage of mites which die during the overall period of development and is given by this formula:

$$M_{total} = \frac{N_{egg} - N_{adult}}{N_{egg}} \times 100\% \tag{18}$$

Table 5 shows the total mortality, in percent, during the development of mites from fresh eggs to adults.

The data is concentrated along the 75% RH row and the 25°C column. The mortality increases at low and high RH, and also at low and high temperatures. The minimum mortality is at 80% RH and 25°C. Two independent quadratic curves are used to fit the RH and the temperature data separately and are then combined together assuming there are no cross correlations.

$$M_{total}(RH,T) = 803.59 - 15.97RH + 0.11RH^2 + 14.14T + 0.27T^2 \tag{19}$$

Figures 8 and 9 shows the data and the fits.

The probability of mortality during the complete development period is equal to the product of the probability of mortality during the egg development phase and the juvenile phase.

$$\frac{M_{total}(RH,T)}{100\%} = \frac{M_{egg}(RH,T)}{100\%} \times \frac{M_{juvenile}(RH,T)}{100\%} \tag{20}$$

Using the same logic from the previous section on the survival rate during the egg development phase, the hourly survival rate of juveniles is given by:

$$S_{juvenile}(RH,T) = \left(1 - \frac{M_{juvenile}(RH,T)}{100\%}\right)^{t_{unit}/D_{juvenile}(RH,T)} \tag{21}$$

Substituting equation 20 into equation 21 gives

$$S_{juvenile}(RH,T) = \left(1 - \frac{M_{total}(RH,T)}{M_{egg}(RH,T)}\right)^{t_{unit}/D_{juvenile}(RH,T)} \tag{22}$$

Survival rate during the adult phase $S_{adult}(RH,T)$

Since no direct or indirect measurements of the survival rates or mortality for adults have been published, we have assumed that the mortality of adults is the same as the mortality of juveniles.

$$M_{adult} = M_{juvenile} = \frac{M_{total}}{M_{egg}} \times 100\% \tag{23}$$

This leads to the rate of adult mites surviving per hour as:

$$S_{adult}(RH,T) = \left(1 - \frac{M_{adult}(RH,T)}{100\%}\right)^{t_{unit}/D_{adult}(RH,T)} \tag{24}$$

Substituting equation 23 into equation 24 gives:

$$S_{adult}(RH,T) = \left(1 - \frac{M_{total}(RH,T)}{M_{egg}(RH,T)}\right)^{t_{unit}/D_{adult}(RH,T)} \tag{25}$$

Adult female egg laying rate $F_{adult}(RH,T)$

The fecundity or total mean number of eggs produced by a female mite during her adult life is given in Table 6 in number of eggs.

A cubic formula for both RH and temperature is used to describe the data,

$$f = 336.71 - 65.08RH + 1.16RH^2 - 0.0064RH^3 + 89.02T - 3.04T^2 + 0.032T^3 \tag{26}$$

Figures 10 and 11 show the data and the fits.

The simplifying assumption has been made that female adults lay eggs at a constant rate throughout their adult lifetime. Therefore the probability that they will lay eggs in any one time slice is given by:

$$F(RH,T) = f \times t_{unit} / D_{adult}(RH,T) \quad (27)$$

Using the POPMITE Simulation

The POPMITE simulation program has been written in PERL (www.perl.com). POPMITE can read the hygrothermal conditions, together with a description of the initial structure of the mite population, and predict the population dynamics.

To illustrate how the model functions, POPMITE has been used to predict the population dynamics of 100 freshly laid eggs (no juveniles or adults at the start) in an arbitrarily chosen constant climate of 75%RH and 35°C for 30 days. Figure 12 shows the results.

The plot shows the number of mites as a function of time. Initially there are 100 eggs at the beginning of the first day. Over the next 4 days (the development period at 35°C) the number of eggs has decreased to roughly 80 % (the survival probability). At this point all the eggs turn into juveniles and the number of eggs drops to zero. After approximately 10 further days the number of juveniles has decreased to 60% approximately to leave about 45 juveniles. At this point the juveniles turn into adults and the number of juveniles drops to zero. Immediately the adults start to lay new eggs. Fresh batches of new eggs are then laid every time slice, depending on the number of adults. The number of adults gradually decreases, according to the survivability rate. However after 4 further days the first of the new eggs to be laid start to hatch into juveniles, and the number of juveniles starts to increase. The number of eggs in the population is then equal to the number of eggs being laid minus the number of eggs dying, minus the number of eggs changing into juveniles, and so the egg population starts to decrease. After a further 10 days the juveniles start to turn into adults, and there is consequently a sharp drop in the number of juveniles, a sharp rise in the number of adults and also a sharp rise in the number of eggs. At 29 days the number of adults drops sharply before the upward trend resumes. These are the adults from the original batch of eggs dying.

If this simulation is left to run for any length of time the population of mites will quickly become enormous, which is to be expected for these relatively favourable conditions, with unlimited food supplies.

Preliminary validation by comparison of POPMITE predictions with measurements of mite population growth.

The DP mite population growth and decline, after a 21 day period, under a range of constant RH and temperature conditions, have been measured as reported by

Crowther et al. (2006). A population growth multiplication factor was calculated for each RH and temperature combination by comparing the final population of juveniles and adults with the initial population of juveniles and adults. The population of eggs was not measured but as the initial populations were mature and well mixed it was assumed that there were a similar number of eggs in each sample. No measurement, other than the combined total number of juveniles and adults, was made of the initial population structure.

POPMITE can be used to predict the measurements of the population growth factor. With no other information on the starting population structure, we assume a population structure of 1:1:1 of eggs, juveniles and adults with a spread over all ages. The predictions and data with statistical error bars from Crowther et al (2006) are shown as a function of RH in figures 13, 14, 15, 16 and 17 for fixed temperatures of 15°C, 20°C, 25°C, 30°C and 35°C respectively. The discontinuity at 60% RH in the POPMITE predictions in figures 13 to 17 are due to the use of the arbitrary step change at 60%, as explained earlier, in equation 10 to describe adult longevity and equations 13 and 14 to describe egg mortality.

The POPMITE model under-predicts the growth factor at high RH when the temperature is 30°C, but otherwise the predictions and measurements show a good agreement with each other over the whole RH and T range.

A similar experiment as described by Crowther et al with more detailed measurements of the initial and final population structures should be able to provide a more comprehensive validation of POPMITE under constant conditions. However the very good agreement with the present data gives confidence in POPMITE and allows for a preliminary validation.

Predicting mite population growth at a range of constant conditions

The POPMITE simulation program can now be used to predict mite population growth or decline for a wide range of constant RH and T combinations. Figure 18 shows the predicted growth of a population consisting of 100 freshly laid eggs after 30 days. The thick contour represents the RH and T combinations where the complete population (eggs, juveniles and adults) is numerically equal to the starting population. Figure 18 show a central 'island' of growth with a maximum growth factor of 11.476 (538.8 Eggs, 553.9 Juveniles and 54.9 Adults) at 29°C and 76%RH. Outside this island the populations are in decline, with a higher decline at higher temperatures.

The discontinuity at 60% RH in Figure 18 is again due to the use of the arbitrary step change at 60% in equation 10 to describe adult longevity and equations 13 and 14 to describe egg mortality.

Figure 18 shows the population growth for only 30 days, which means that for populations at low temperatures the mites have not yet completed a full life cycle. At temperatures below 13°C eggs will not have had time to hatch into juveniles, and below about 24°C juveniles will not have had time to turn into adults. The lower

bound of the 'island' of growth at 24°C as shown in Figure 18 is due to mites not reaching maturity and therefore being unable to produce eggs to increase the population.

Figure 19 shows the predicted growth of a population consisting of 100 freshly laid eggs after 60 days. The maximum growth factor of 365.293 (20,002.9 Eggs, 13,062.7 Juveniles and 3,463.7 Adults) is now at 31°C and 76%RH, which is at a slightly higher temperature than the predicted maximum of 29°C for the 30 day simulations. It is also massively increased. Mites held in these conditions are unlikely to reach such numbers unless there is an unlimited amount of food and space available to them. Wilkinson et al. (2002) have shown that 1 gram of food is enough for a population of about 12,000 mites (juveniles and adults) to develop after 18 weeks from only 2 mating pairs held at 25°C and 75%RH. The population then gradually declined as the food was consumed. A limit on the population growth based on the available food can be incorporated into POPMITE if needed. This will be the subject of a future paper by the authors.

The area over which the population is either stable or growing is now much larger. However the lower bound of this growth 'island' is again due to the fact that below 18°C juveniles will not have been able to turn into adults.

The total mortality as described by equation 19 and shown in Figure 8 has a minimum at 26°C and the fecundity as described by equation 26 and Figure 11 has a maximum at 23°C. This would suggest that the maximum growth factor should be found at much lower temperatures than the 29°C for 30 days and 31°C for 60 days. The reason for this apparent discrepancy is that even though the conditions are less favourable, the development rate is very much increased at higher temperatures. The mites are therefore able to replenish their numbers at a faster rate than mites held at lower temperatures.

It is interesting to note that the models based on the constant growth multiplication factor (Crowther et al. 2006) would have predicted a different growth factor after 60 days given the growth after 30 days. Assuming that the growth was constant over the first 30 days then the growth multiplication factor per day would have been $11.476^{(1/30)} = 1.085$. In other words if there where 100 mites then at the end of first day there will be 108.5 mites and at the end second day will be 117.2 mites and so on. This model then predicts a population growth of only $1.085^{60} = 131.699$ after 60 days compared to the POPMITE prediction of 365.293. This illustrates the different results that can be obtained when population structure is taken into account.

Simulation using changing hygrothermal conditions

Once it has been further validated, the model can be used to predict the population dynamics of mites held in constant hygrothermal conditions. Unfortunately the hygrothermal conditions of the typical habitats of mites, such as a mattress, can vary

dramatically. Ridley et al. (2006) have recorded the micro-climatic conditions found throughout the beds of a number of people over a period of a few days. These measurements show that when the bed is empty the mattress is in climatic equilibrium with the bedroom. However once the bed is occupied there is a dramatic increase in the absolute humidity and temperature in the mattress immediately under the body. The temperature increase is usually so high that the corresponding RH drops. Even though the absolute humidity in the bed has increased due to the presence of a sweating, moisture breathing sleeping person, the excess vapour is not enough to increase the relative humidity, since the temperature has risen by a disproportionately large amount at the same time, thus resulting in a drop in relative humidity. For a discussion on this point see Pretlove et al. (2005)

POPMITE can accept continually changing conditions as input, although the quality of the prediction is likely to be degraded the greater the transient nature of the hygrothermal conditions. The reason for this is that at present the model assumes that the survival rates for each phase are constant; in other words the probability of surviving in one time slice does not depend on how long that batch of mites has been exposed to the current environment.

De Boer et al. (1997, 1998) and Pike et al. (2005) have shown that DP populations held in unfavourable conditions of low RH, but given brief periods of favourable high RH conditions every day, are able to survive and even grow. Mite populations held continuously in harsh conditions decline and eventually die out. This suggests that individual mites have a survival probability which depends on how long they are held in harsh conditions.

Arlian and Wharton (1974), Arlian (1975, 1992), Wharton (1978), Arlian and Veselica (1979, 1981a,b) and de Boer (2000) have investigated the water balance of mites (DP and *Dermatophagoides farinae*) and have developed equations which describe the water content of a mite under different hygrothermal conditions. In low RH conditions mites initially lose moisture very quickly, but after a short period of time are able to reduce the water loss drastically, enabling them to survive for an extended period of time. In high RH conditions, mites are able to recover any lost moisture very quickly and to maintain it at high levels to allow them to function and reproduce.

An improvement to the POPMITE model is therefore being developed which will include parameters to track the water contents of mites and therefore reproduce the correct response to conditions fluctuating between high and low RH. Until this is available a minimum survival rate is applied to all batches to allow at least some mites to survive unfavourable conditions. However the resultant predictions are unlikely to be accurate in very transient conditions.

Conclusions

A generalised model of the life cycle of a house dust mite is presented. Previously published data on the physiological effects of exposure of the DP mite to constant

relative humidity and temperature combinations is used to construct a model and computer simulation called POPMITE. For a given starting population structure and climate history, POPMITE is able to predict the most likely population dynamics for the house dust mite DP.

Preliminary validation by comparisons of the POPMITE predictions with measurements of population growth at constant hygrothermal conditions shows good agreement.

Contour plots showing the predicted growth of a population consisting of 100 freshly laid eggs after 30 days and 60 days in a range of RH and T combinations show that temperature plays an important role in controlling the growth and decline of populations.

POPMITE produces very different predictions from models based on the assumption of a constant growth multiplication factor, which are the basis of the models of Cunningham and Crowther. These models are simple and easy to use and can gave a broad indication of the population growth, but they will never be able to give as accurate a prediction as POPMITE, since the population structure and therefore the population growth multiplication factor changes all the time, even under constant conditions.

The current version of POPMITE, when fully validated, will give good predictions of house dust mite population dynamics where conditions are constant or changing slowly, but will tend to give degraded predictions once conditions start to fluctuate. This is still a significant improvement on the models of Cunningham and Crowther, and POPMITE can already be used as part of a study to test scenarios for changing building design or occupant behaviour to reduce mite populations in beds.

A study to provide a precise and comprehensive dataset of the physiological response of DP to a wide range of both constant and varying environmental conditions and diets is under way. This dataset is required to calibrate the parameters needed to track the moisture content of mites for a more accurate version of the POPMITE simulation that will be able to cope fully with varying hygrothermal conditions.

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References

Andersen, A (1988) Population growth and developmental stages of the house dust mite, *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). J. Med Entomol. 25: 370-373

Arlian LG (1975) Dehydration and survival of the European house dust mite, *Dermatophagoides pteronyssinus*. Journal of Medical Entomology, 12/4: 437-442

Arlian LG (1992) Water balance and humidity requirements of house dust mites. Experimental & Applied Acarology, 16: 15-35

Arlian LG, Rapp CM and Ahmed SG (1990) Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Journal of Medical Entomology, 27/6: 1035-1040

Arlian LG and Veselica MM (1979) Water balance in insects and mites. Comp Biochem Physiol, 64A: 191-200

Arlian LG and Veselica MM (1981a) Effect of temperature on the equilibrium body water mass in the mite Dermatophagoides farinae. Physiology Zoology, 54/4: 393-399

Arlian LG and Veselica MM (1981b) Re-evaluation of the humidity requirements of the house dust mite *Dermatophagoides farinae* (Acari: Pyroglyphidae). Journal of Medical Entomology, 18/4: 351-352

Arlian LG and Wharton GW (1974) Kinetics of active and passive components of water exchange between the air and a mite, *Dermatophagoides farinae*. Journal of Insect Physiology, 20: 1063-1077

Blythe, M.E (1976) Some aspects of the ecological study of the house dust mite. Brit. J. Dis. Chest 70: 3-31.

Boer R de (2000) Rates of water vapour uptake and loss by the house dust mite, Dermatophagoides pteronyssinus (Acari: Pyroglyphidae). Proceedings of Experimental & Applied Entomology, 11: 73-76

Boer R de and Kuller K (1997) Mattresses as a winter refuge for house-dust mite populations. Allergy, 52: 299-305

Boer R de, Kuller K and Kahl O (1998) Water balance of Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) maintained by brief daily spells of elevated air humidity. Journal of Medical Entomology, 35/6: 905-910

Bronswijk JE van (1981) House Dust Biology for Allergists, Acarologists and Mycologists. NIB Publishers, The Netherlands

Case T J (2000) An Illustrated Guide to Theoretical Ecology. Oxford Univ Press. New York.

Colloff, M. J (1987a) Effects of temperature and relative humidity on development times and mortality of eggs from laboratory and wild populations of the European house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae). Experimental and Applied Acarology 3 : 279-289.

Colloff, M. J (1987b) Differences in development time, mortality and water loss between eggs from laboratory and wild populations of *Dermatophagoides pteronyssinus*. Experimental and Applied Acarology 3 : 191-200.

Colloff M (1998) Taxonomy and identification of dust mites. Allergy 53 Supplement 48 : 7-12

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S and Ridley I (2006). A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Experimental and Applied Acarology, 79(2): 127-148

Cunningham MJ (2000) A proposed experimental programme towards control of dust mites by microclimate modification, in Mites, Asthma and Domestic Design III (Eds: Siebers R, Cunningham M, Fitzharris P and Crane J). Wellington Asthma Research Group, Department of Medicine, Otago University, 2000

Dobson R.M (1979) Some effects of microclimate on the longevity and development of *Dermatophagoides pteronyssinus* (Trouessart). Acarologia 21: 482-486.

Ford AW, Rawle FC, Lind P, Spieksma FTM, Lowenstein H and Platts-Mills TAE (1985) Standardization of *Dermatophagoides pteronyssinus*: Assessment of potency and allergen content in ten coded extracts. International Archives of Allergy and Applied Immunology 76: 58-67.

Gamal-Eddin FM, Abou-Sinna FM, Tayel SE, Aboul-Atta AM, Seif AM, Gaafar SM (1983a). Duration of the developmental stages of house dust mites *Dermatophagoides farinae* and DP under controlled temperatures and relative humidities to pave the way in from of the workers in the field of house dust mite bronchial asthma. 1. Pre-imaginal period. Journal of the Egyptian Society of Parasitology 13 : 319-334.

Gamal-Eddin FM, Shehata KK, Tayel SE, Abou-Sinna FM, Aboul-Atta AM, Seif AI, Imam MH, Hafez AH (1983b). Duration and the development stages of house-dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled conditions to pave the way in front of the workers in the field of house-dust mite asthmatic bronchitis. 2. Oviposition period, fecundity and oval duration. Journal of the Egyptian Society of Parasitology, 13: 557-581

Gamal-Eddin FM, Shehata KK, Abou-Sinn FM, Tayel SE, Aboul-Atta AM, El-Ahmedaawy BA, Fayed MA, Hafez AH and Imam MH (1983c) Duration of the developmental stages of house dust mites DF and DP under controlled conditions to pave the way in from of the workers in the field of house dust mite asthmatic bronchitis 3. Larval Duration. Journal of the Egyptian Society of Parasitology 13 : 583 – 595

Hart, BJ. and Fain, A (1988) Morphological and biological studies of medically important house-dust mites. Acarologia 29: 285-295.

Ho, TM. and Nadchatram, M (1984) Life cycle and longevity of *Dermatophagoides pteronyssinus* (Trouessart) (Acarina: Astigmata: Pyroglyphidae) in a tropical laboratory. Trop. Biomed 1: 159-162.

Hodgson, R.K (1976) Sex ratio and sex determination in the American house dust mite, Dermatophagoides farinae. Annals Ent. Soc America, 69: 1085-6.

Nisbet, RM. and Gurney, WSC. (1982) Modelling Fluctuating Populations. John Wiley and Sons, Chichester.

Leslie, PH. (1945) On the use of matrices in certain population mathematics. Biometrika, 35: 183-212.

Pretlove S, Oreszczyn T, Ridley I, Wilkinson T and Crowther D (2005) A steady state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements. Building Services Engineering Research & Technology, 26/4: 301-314

Pike A, Cunningham M and Lester P (2005) Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) at constant and simultaneously fluctuating temperature and humidity conditions. Journal of Medical Entomology, 42/3: 266-269

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M and Hart B (2006) A model to predict the hygrothermal conditions experienced by house dust mites within beds. Submitted to Indoor Air.

Spieksma, F. Th M (1967) The House Dust Mite *Dermatophagoides pteronyssinus* (Trouessart, 1897). PhD Thesis, Univ. of Leiden, 65 pp.

U.S. Department of Energy (US-DOE) (2004) EnergyPlus Energy Simulation Software. http://www.eere.energy.gov/buildings/energyplus

Voorhorst R, Spieksma F and Varekamp H (1969) House Dust Atopy and the House Dust Mite, *Dermatophagoides pteronyssinus* (Trouessart, 1897). Stafleu's Scientific Publishing Company, Leiden

Wharton G (1978) Uptake of water vapour by mites and mechanisms utilized by the Acaridei. In Comparative Physiology: Water, Ions and Fluid Mechanics (Eds: Schmidt-Nielsen K, Bolis L and Maddrell S). Cambridge University Press, Cambridge.

Wilkinson T, Horwood J, Cox P, Crowther D, Ridley I, Pretlove S and Oreszczyn T (2002) Factors affecting the carrying capacity (K) of a mattress for the house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) 11th International Congress of Acarology. Universidad Nacional Autonoma de Mexico, Merida, Yucatan, Mexico, 8th-13th September 2002

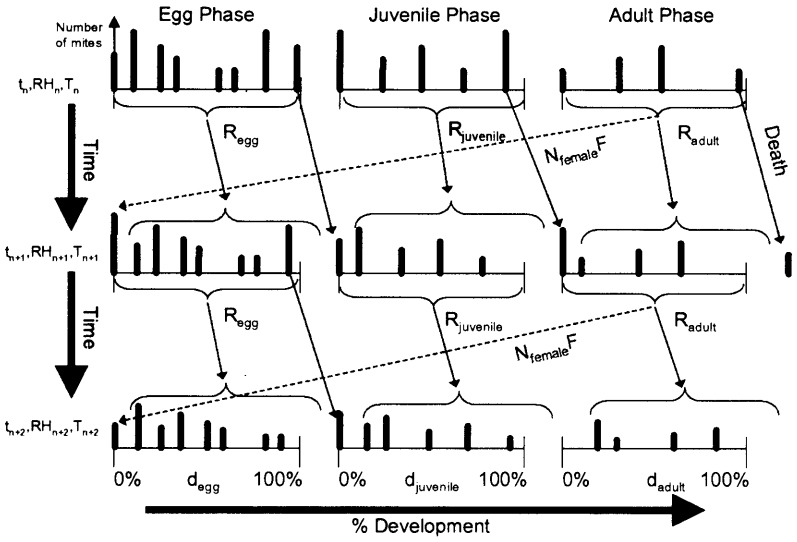

Figure 1 Schematic diagram of a simplified mite life cycle

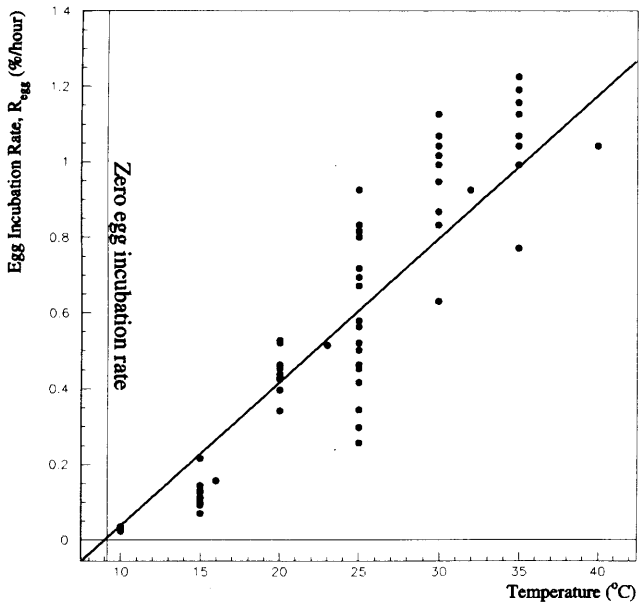

Figure 2 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite egg development rate as a function of temperature

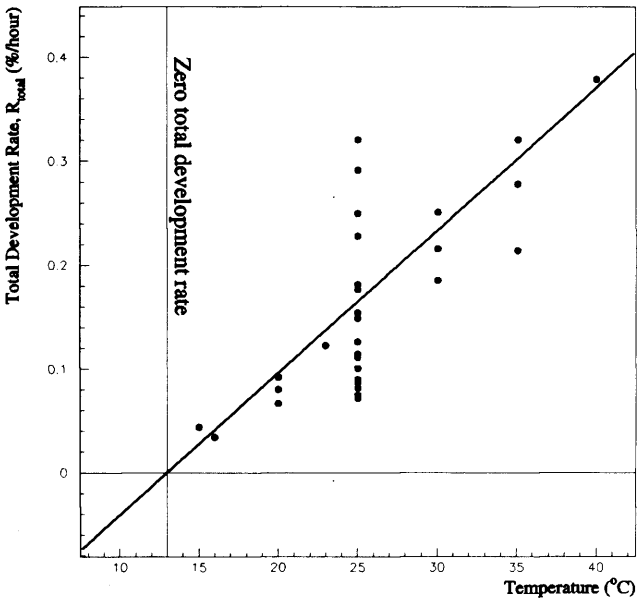

Figure 3 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite total development rate (from egg to adult) as a function of temperature

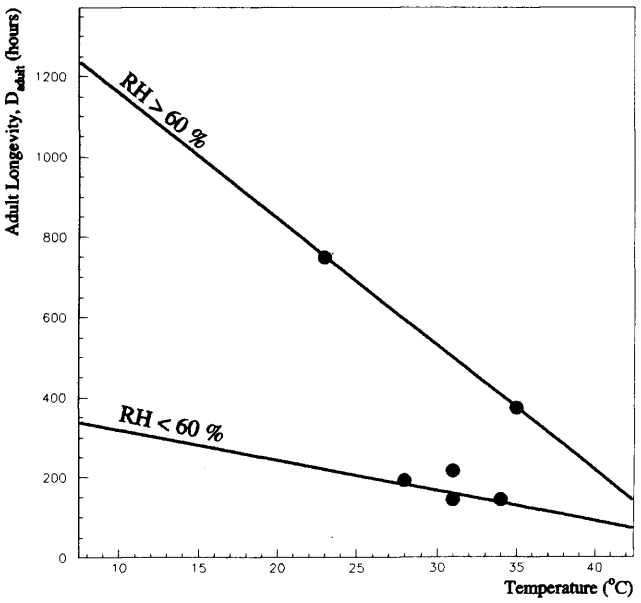

Figure 4 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite adult longevity as a function of temperature

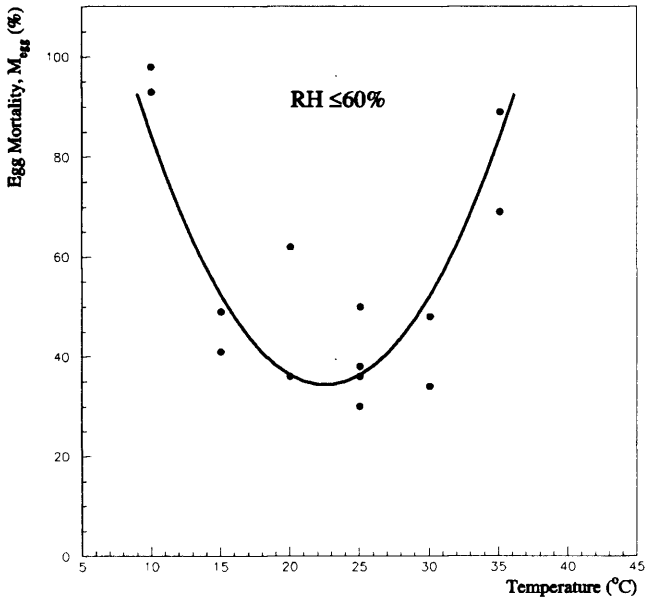

Figure 5 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite egg mortality as a function of temperature for relative humidity values less than 60%

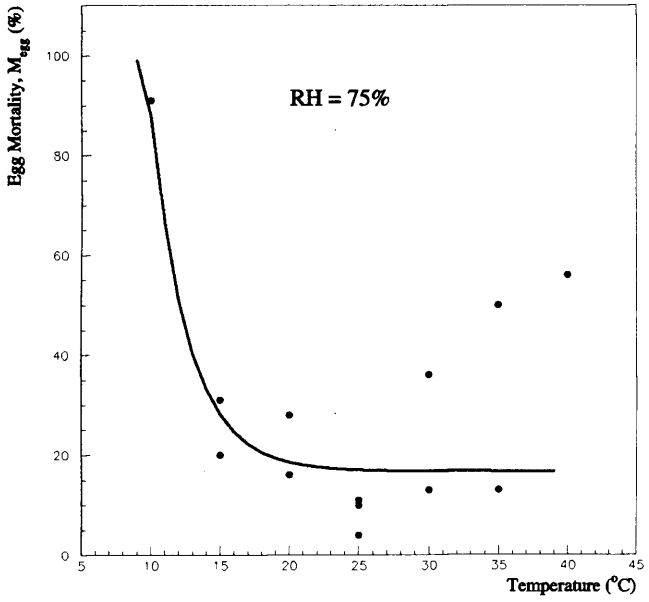

Figure 6 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite egg mortality as a function of temperature at a fixed relative humidity of 75%

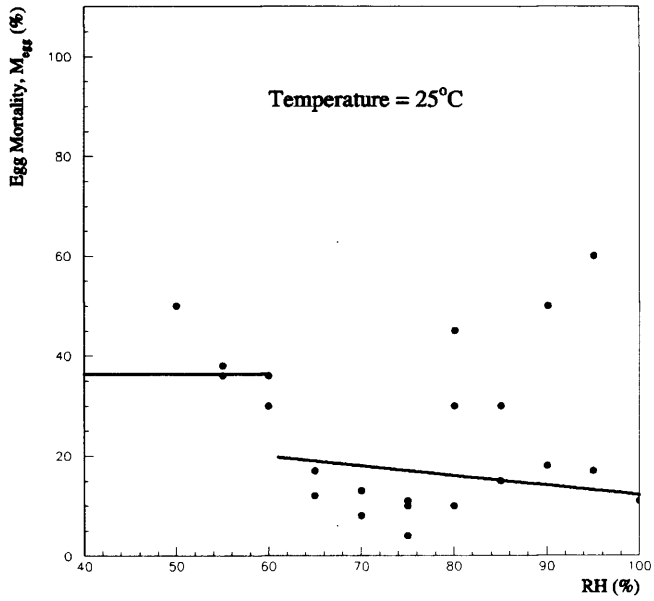

Figure 7 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite egg mortality as a function of relative humidity at a fixed temperature of 25°C

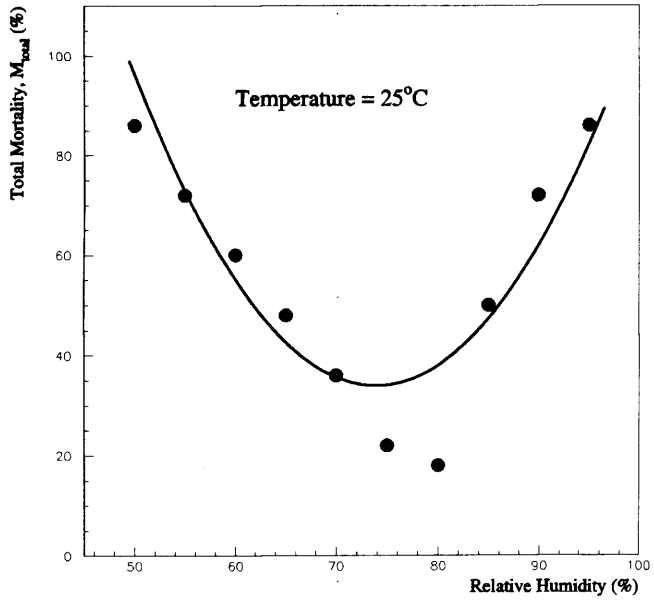

Figure 8 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite total mortality, from egg to adult, as a function of relative humidity for a fixed temperature of 25°C

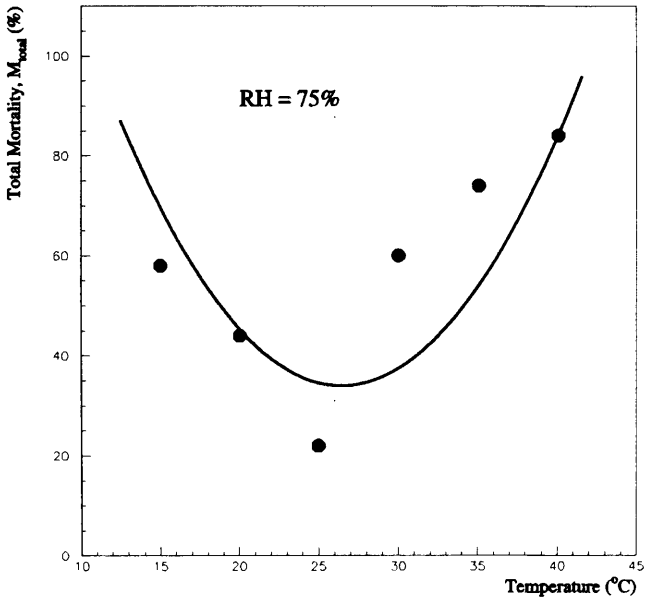

Figure 9 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite total mortality, from egg to adult, as a function of Temperature for a fixed relative humidity of 75%

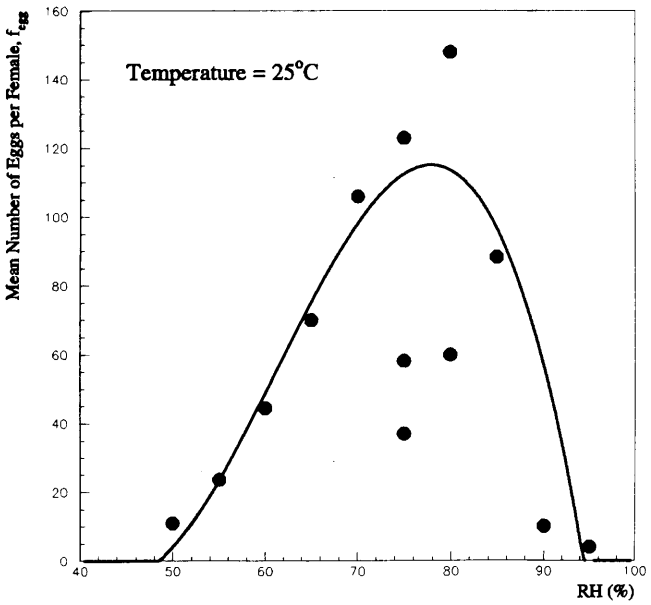

Figure 10 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite mean number of eggs per female during adult life as a function of relative humidity at a fixed temperature of 25°C

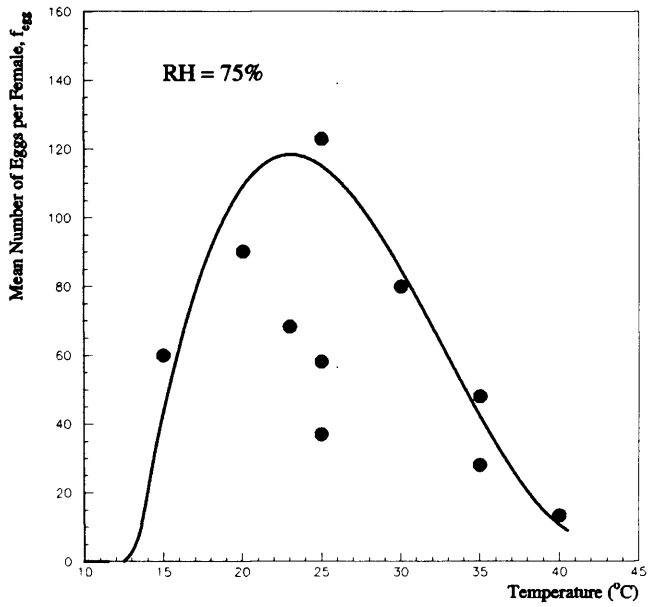

Figure 11 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite mean number of eggs per female during adult life as a function of Temperature at a fixed relative humidity of 75%

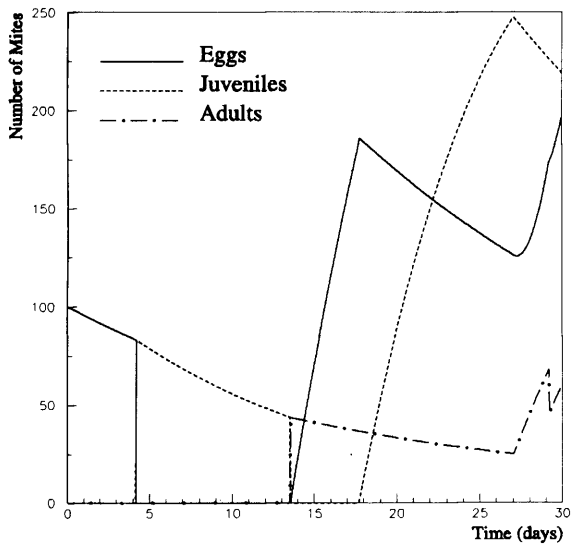

Figure 12 Prediction of the population dynamics of a batch of 100 freshly laid eggs held at a constant temperature of 35°C and a constant relative humidity of 75%. The total number of eggs in all batches is plotted as a solid line, the total number of juveniles as a dashed line and the total number of adults as a dash-dotted line

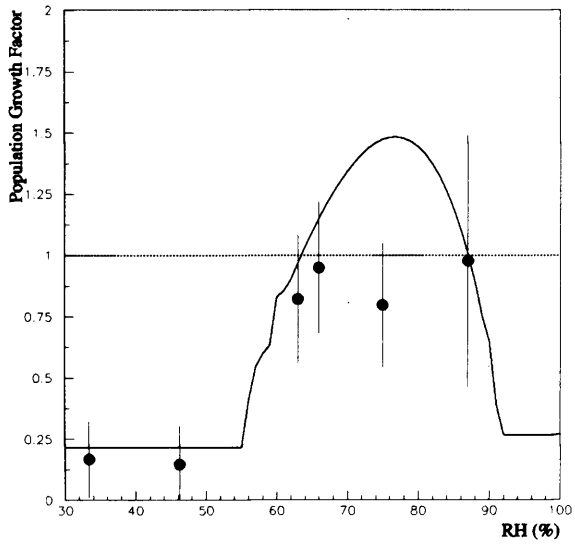

Figure 13 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) for a range of RH values at a fixed temperature of 15°C.

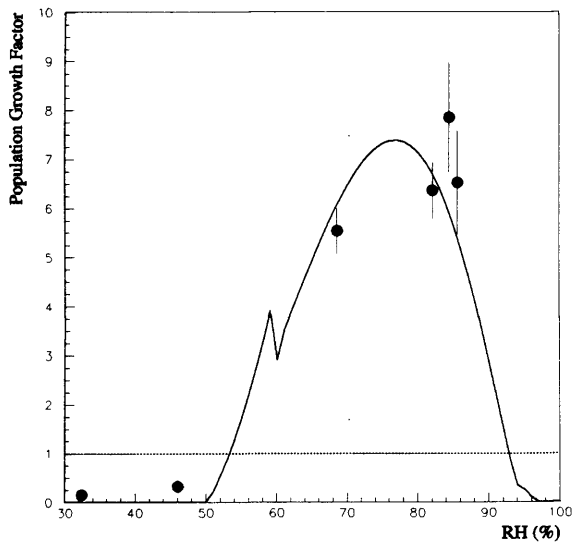

Figure 14 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae)for a range of RH values at a fixed temperature of 20°C.

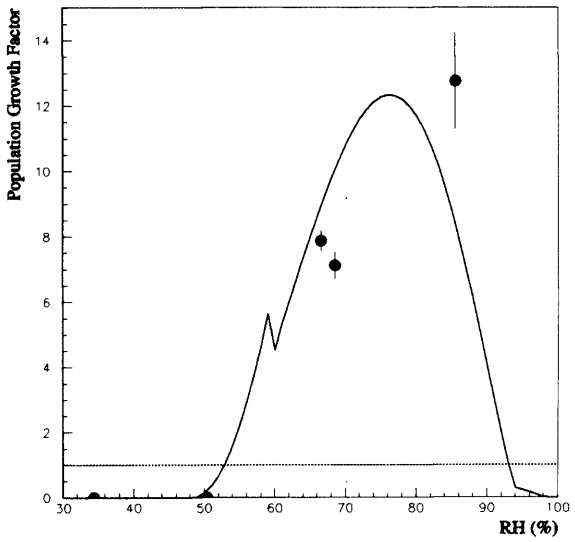

Figure 15 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae)for a range of RH values at a fixed temperature of 25°C.

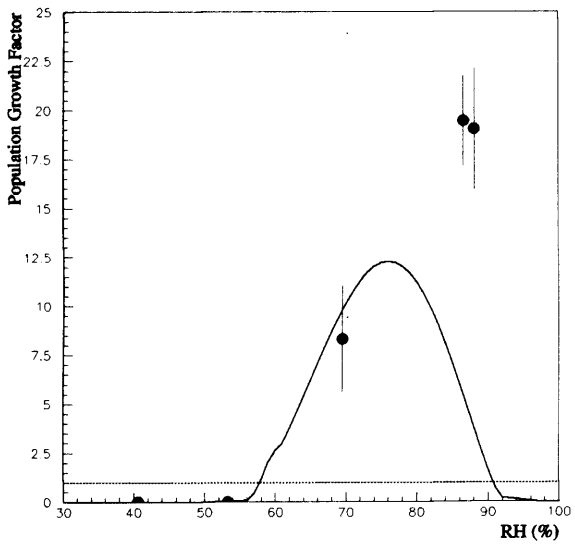

Figure 16 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae)for a range of RH values at a fixed temperature of 30°C.

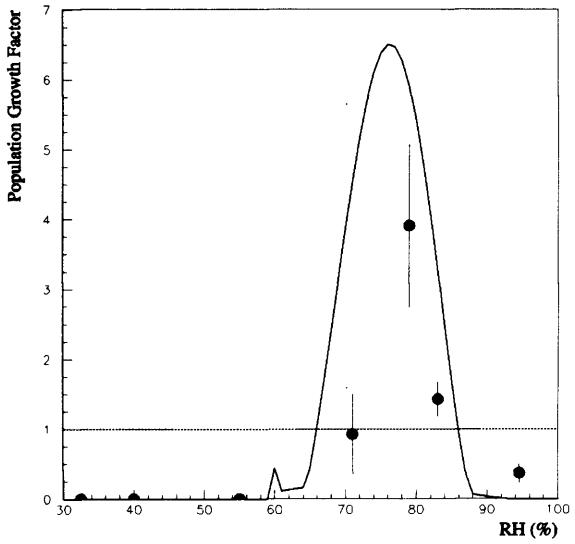

Figure 17 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae)for a range of RH values at a fixed temperature of 35°C.

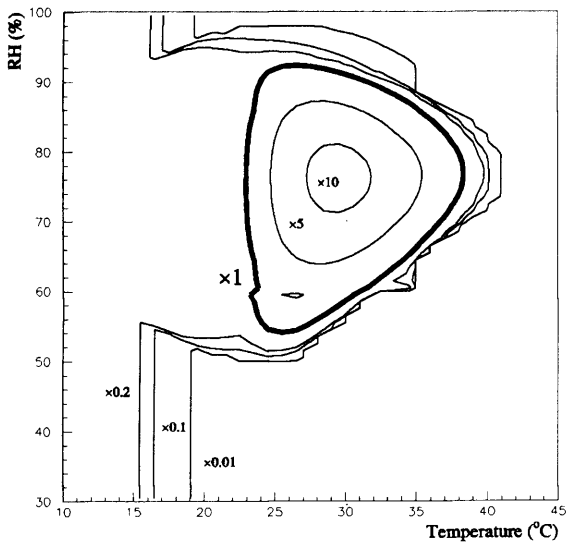

Figure 18 A contour plot of the complete mite population (eggs, juveniles and adults) growth factor after 30 days as predicted with the POPMITE model starting with 100 freshly laid eggs, for a range of relative humidity and temperature combinations

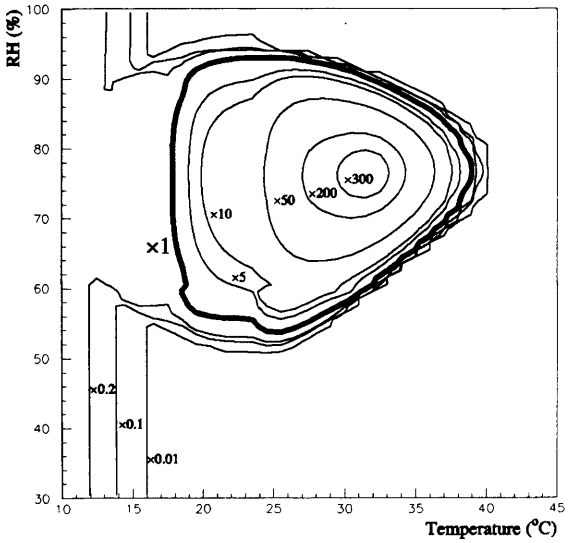

Figure 19 A contour plot of the complete mite population (eggs, juveniles and adults) growth factor after 60 days as predicted with the POPMITE model starting with 100 freshly laid eggs, for a range of relative humidity and temperature combinations

Table 1 Development duration for *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite eggs in days, [a]Colloff (1987a, 1987b), [b]Gamal Eddin et al. (1983b), [c]Arlian et al. (1990), [d]Spieksma (1967)

	Temperature(°C)																
RH(%)	10	15		16	20		23	25			30		32	35			40
50									16.2[b]								
55	180[a]	59[a]			9.7[a]			6.2[a]	14[b]		5[a]			4.2[a]			
60	155[a]	42[a]			9.2[a]			6[a]	12.1[b]		4.8[a]			4.2[a]			
65	120[a]	37[a]			9.1[a]			5.8[a]	10[b]		4.4[a]			3.9[a]			
70	135[a]	29[a]			7.9[a]			5.1[a]	9.2[b]		4.1[a]			4[a]			
75	145[a]	32[a]	19.3[b]	26.6[c]	7.9[a]	12.2[b]	8.1[c]	4.5[a]	8.3[b]		3.9[a]	6.6[b]	4.5[c]	3.9[a]	5.4[b]	3.9[c]	4[b]
80	150[a]	33[a]			8[a]			5[a]	7.2[b]	6[d]	3.7[a]			3.7[a]			
85	160[a]	38[a]			9[a]			5.2[a]	8[b]		4[a]			3.6[a]			
90	150[a]	42[a]			9.8[a]			9[a]	10[b]		4[a]			3.5[a]			
95	150[a]	44[a]			9.5[a]			5[a]	14[b]		4.1[a]			3.4[a]			
100	175[a]	45[a]			10.5[a]			5.2[a]			4.2[a]			3.9[a]			

Table 2 Total development time (days) of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mites from egg to adult, [a]Gamal Eddin et al (1983a), [b]Arlian et al. (1990) , [c]Arlian (1975), [d]Dobson (1979) , [e]Hart and Fain (1988), [f]Blythe (1976), [g]Anderson (1988), [h]Ho and Nadchatram (1984), [i]Spieksma (1967)

	Temperature(°C)															
RH(%)	15	16	20		23	25						30		35		40
50						58[a]										
55						51[a]										
60						46.5[a]										
65						41.5[a]										
70						37.5[a]										
75	95[a]	123[b]	62.4[c]	52[a]	34[b]	36.5[a]	14.3[c]	14.3[e]	33[f]	28[h]		19.3[b]	22.5[a]	15[b]	19.5[a]	11[a]
80			45.3[c]	45.2[d]		27[a]	18.3[c]	16.7[d]	13[d]	23.6[g]	23[i]	16.6[d]		13[d]		
85						36.5[a]										
90						48[a]										
95						56[a]										

Table 3 Adult longevity of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mites (days), [a]Arlian (1975) and [b]Arlian et al. (1990).

	Temperature(°C)				
RH(%)	23	28	31	34	35
40		8[a]	6[a]	6[a]	
50			9[a]	6[a]	
75	31.2[b]				15.5[b]

Table 4 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) Egg mortality % death at the end of the egg phase, [a]Colloff (1987a, 1987b), [b]Gamal Eddin et al (1983b), [c]Spieksma (1967)

	Temperature(°C)												
RH (%)	10	15		20		25			30		35		40
50								50[b]					
55	93[a]	49[a]		62[a]		36[a]		38[b]	48[a]		89[a]		
60	98[a]	41[a]		36[a]		36[a]		30[b]	34[a]		69[a]		
65	81[a]	37[a]		26[a]		17[a]		12[b]	15[a]		32[a]		
70	92[a]	35[a]		13[a]		13[a]		8[b]	10[a]		24[a]		
75	91[a]	31[a]	20[b]	28[a]	16[b]	11[a]	10[b]	4[b]	13[a]	36[b]	13[a]	50[b]	56[b]
80	90[a]	17[a]		14[a]		30[a]	45[c]	10[b]	26[a]		5[a]		
85	79[a]	21[a]		24[a]		15[a]		30[b]	8[a]		5[a]		
90	84[a]	18[a]		14[a]		18[a]		50[b]	16[a]		10[a]		
95	85[a]	23[a]		16[a]		17[a]		60[b]	14[a]		13[a]		
100	90[a]	26[a]		19[a]		11[a]			7[a]		15[a]		

Table 5 Total egg to adult mortality of the *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite, Gamal Eddin et al. (1983a)

	Temperature(°C)					
RH(%)	15	20	25	30	35	40
50			86			
55			72			
60			60			
65			48			
70			36			
75	58	44	22	60	74	84
80			18			
85			50			
90			72			
95			86			

Table 6 Mean number of eggs produced by a female *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite during her adult life, [a]Gamal Eddin et al. (1983b), [b]Arlian et al. (1990) , [c]Hart and Fain (1988), [d]Spieksma (1967).

	Temperature(°C)								
RH (%)	15	20	23	25		30	35		40
50				11[a]					
55				23.7[a]					
60				44.5[a]					
65				70[a]					
70				106[a]					
75	60[a]	90.2[a]	68.4[b]	123[a]	58.2[c]	80[a]	28[a]	48[b]	13.3[a]
80				148[a]	60[d]				
85				88.4[a]					
90				10[a]					
95				4[a]					

Appendix A.0: Published Papers

Published: Journal of Medical Entomology, 44(4): 568-574.

A.0.3: *Reproduction and development of laboratory and wild house dust mites (Dermatophagoides pteronyssinus (Acari: Pyroglyphidae)) and their relation to the natural dust ecosystem*

B.J. Hart[1], D. Crowther[2], T. Wilkinson[2,3], P. Biddulph[2,4], **M. Ucci**[4], S. Pretlove[5], I. Ridley[4] And T. Oreszczyn[4]

[1]Royal Agricultural College, Stroud Road, Cirencester, Gloucestershire GL7 6JS, UK
[2]Martin Centre, Cambridge University, 1 Scroope Terrace, Cambridge CB2 1PX, UK
[3]Medical Entomology Centre, Barrington Road, Shepreth, Royston SG8 6QZ, UK
[4]Bartlett School of Graduate Studies, University College London WC1E 6BT, UK
[5]School of Architecture & Landscape, Kingston University, London KT1 2QJ, UK

Abstract

Life histories of 'wild' house dust mites (*Dermatophagoides pteronyssinus* (Trouessart)) were compared to laboratory cultures, using a diet consisting of skin and dust or a laboratory diet of dried liver and yeast. Under constant conditions of 25°C, 75% relative humidity (RH), fecundity and rate of reproduction were higher in laboratory cultures on both diets compared to wild mites. There were also trends for a shorter pre-reproductive period and more rapid egg development of laboratory mites compared to wild mites. Overall there was little effect of diet on either strain of mites at 75% RH. At low RH (64%), fecundity was significantly lower (for both strains on both diets) and there were also trends for longer pre-reproductive period, reduced rate of reproduction, reduced adult survival and prolonged egg/juvenile development compared to 75% RH. Additionally egg and juvenile mortality were significantly higher on the liver/yeast diet. Overall the skin/dust diet favoured both strains of mites at 64% RH. On the liver/yeast diet at 64% RH wild mite adults performed significantly better than laboratory mites and egg mortality was lower. These results suggest that laboratory mites have stronger reproduction and development than wild mites, except when under environmental stress, and that diet is a significant factor, particularly in sub-optimal conditions. This could have important implications for predictive models of house dust mite populations in their natural habitat. Ideally, such models should be developed using data from wild dust mite populations reared on a natural diet.

Key Words:

Dermatophagoides pteronyssinus, wild populations, life history.

INTRODUCTION

Population models to predict house dust mite populations in the home are currently under development (Pretlove *et al.* 2001, 2005; Crowther *et al.* 2006, Biddulph *et al.* 2007). Their aim is to assist in the effective control of mites by manipulating the temperature and relative humidity in their habitats, psychrometric conditions being known to play a crucial role in their survival (Cunningham 1999, Pretlove *et al.* 2002). The models set out to simulate, first, psychrometric conditions in mite habitats (given climate and building characteristics) and, second, the effect of these conditions on house dust mite populations. In this way the most successful and feasible strategies for achieving psychrometric control can be determined, whether by improving ventilation or by a combination of modifications to building design, building operation and occupant behaviour (e.g. with respect to moisture production, window opening habits, etc.). However, the population models upon which these simulations depend require mite physiology data inputs which relate to the house dust ecosystem.

Data on house dust mite reproduction and development have, until now, predominantly been obtained from mite cultures that have been reared for many years in laboratory conditions (Spieksma 1967, Blythe 1976, Dobson 1979, Gamal-Eddin *et al.* 1983a, 1983b, 1983c; Ho and Nadchatram 1984, Andersen 1988, Hart and Fain 1988, Arlian *et al.* 1990). However, in 1987, Colloff (1987a, 1987b) studied eggs from wild populations of house dust mites and suggested that they differed from eggs from laboratory populations with respect to their development time, mortality and water loss. There have been no subsequent studies on wild populations of house dust mites and no data are available on juvenile or adult physiology from wild cultures.

The principal aims of this study were therefore to obtain more detailed information on the physiology of wild house dust mite populations (*Dermatophagoides pteronyssinus* (Trouessart) compared to laboratory populations and to determine the importance of wild mite data for predictive mite

population models compared to existing data from long-term laboratory populations.

MATERIALS AND METHODS

Mite Cultures.

The laboratory strain of *D. pteronyssinus* had been reared for at least 10 years under constant laboratory conditions of 25°C temperature and 75% relative humidity (RH). Prior to experiments they were reared under these constant hygrothermal conditions on a typical optimised liver/yeast diet of ground dried porcine liver (Oxoid, UK) and brewers yeast (Holland and Barrett UK 1:1 (w:w)).

A 'wild' strain of *D. pteronyssinus* was collected from carpet dust from a UK home in September 2004. From the time of collection, this culture was reared under fluctuating hygrothermal conditions, that is to say fluctuating room temperatures and a diurnal RH fluctuation of 8h at 64% RH and 16h at 75% RH. Wild cultures were reared on a mixture of 1:0.1 (w:w) house dust and non-degreased (fresh) skin scales, with no addition of yeast. Experiments using these wild cultures were started in July 2005.

Mite Physiology Studies.

Glass micro-culture vials 12 mm diameter x 10 mm depth were used to hold individual couples (males attached to tritonymphs) isolated from the laboratory or wild cultures for determination of adult survival and reproduction. Glue was applied around the rim of the vials to prevent escape of the mites and an equal quantity of food was added to each vial. 10 couples were used for each assay and initially observations were made daily to determine pre-reproductive period and then 2-3 times weekly for further egg production and adult survival.

The liver/yeast diet (described above) and a skin/dust diet were used in separate experiments to determine the influence of diet on mite performance. To

standardise the skin/dust diet, a stock of mattress dust was collected from the beds of a total of 20 non-smokers and pooled. It was then frozen at –20°C for a minimum of one week to kill any mites, sieved through a 500 micron mesh and then kept at room temperature for at least one month before use in experiments. The aim of the latter step was to enable recovery of house dust fungi after the freezing step. A pooled stock of skin scales was obtained using beard shavings collected from electric razors of 8 volunteers and were left untreated at room temperature before adding to the dust stock at the start of each experiment to provide a 1:1 (w:w) mixture. No yeast was added to this 'natural' skin/dust diet.

To obtain eggs for development studies, thirty adult females were added to glass micro-culture vials as described above containing either the liver/yeast diet or the pooled skin/dust diet. They were left at 25°C, 75% RH until 50 eggs were laid, the females were then removed and the eggs placed into the relevant hygrothermal conditions for the experiment. Observations were made daily for egg hatching and 2-3 times weekly for juvenile mortality and development.

Constant hygrothermal conditions of 25°C and 75% RH or 64% RH were used in separate experiments to represent optimal laboratory rearing conditions and the lower RH typical of a domestic environment respectively. RH was controlled inside airtight plastic boxes using saturated inorganic salt solutions (Winston & Bates 1960) and was verified periodically throughout experiments using an RH meter.

Principal components analysis (PCA, Legendre and Legendre, 1998) was implemented to assess correlations between response variables, and to test for significant effects of predictor variables on the principal components, thus avoiding inflation of type 1 errors. Provided the PCA showed a response variable was significantly affected by the treatment, it was assessed individually using analysis of variance (ANOVA, Sokal and Rohlf, 1995). Data were log-transformed to meet the assumptions of parametric tests: examination of residuals and fitted values showed that transformation was adequate to remove heteroscedasticity and non-normality of error variance. Significance was assumed at the 5% level ($P = 0.05$), and a Gaussian error distribution was used.

RESULTS

Adults

In the PCA, the first two principal components had eigenvalues greater than one, and together captured 72% of the total variation in the response variables. Principal component 1 (PC1) was positively correlated with reproductive period, female survival, fecundity and male survival, while PC2 was positively correlated with pre-reproductive period and negatively correlated with reproductive rate. The pattern of high factor loadings on the same components suggests that the dependent variables are highly inter-correlated, and likely to show similar patterns among the ANOVAs.

The three-way interaction between strain, diet and RH was a significant predictor of PC1 ($F = 68.1$; $df = 7, 71$; $P < 0.001$). All three manipulated variables were significant predictors of PC2, and the interaction between strain and RH was also significant ($F = 19.8$; $df = 4, 74$; $P < 0.001$). This demonstrates that the effects detected within each life history trait below were real, and not artefacts of accepting random patterns as significant due to the number of separate tests done.

Fecundity.

The three-way interaction between strain, diet and RH was significant ($F = 130.9$; $df = 7, 72$; $P < 0.001$). Fecundity was always higher at 75% compared to 64% RH for all strain and diet combinations (Tables 1 and 2). This was particularly striking on the liver/yeast diet at 64% RH, where fecundity was up to 25 times lower than at 75% RH and up to 10 times lower than on the skin/dust diet at 64% RH.

At 75% RH (Table 1) there was no effect of diet on fecundity, but the laboratory strain of mites had significantly higher fecundity than the wild strain on both diets ($F = 15.6$; $df = 1, 38$; $P < 0.001$).

At 64% RH (Table 2) the interaction between strain and diet was significant ($F = 86.0$; $df = 3, 36$; $P < 0.001$). Both mite strains had higher fecundity on the

skin/dust diet than on the liver/yeast diet, but on the skin/dust diet the laboratory strain had the highest fecundity, while on the liver/yeast diet the wild strain had higher fecundity.

Pre-reproductive Period.

The three-way interaction between strain, diet and RH was significant ($F = 27.9$; $df = 7, 72$; $P < 0.001$). Pre-reproductive period (defined here as the period between mating of female tritonymphs with males and production of first eggs) was shorter at 75% RH than at 64% RH for every combination of strain/diet (Tables 1 and 2). High RH shortened pre-reproductive period to less than any group at low RH, except for wild mites on the skin/dust diet.

At 75% RH (Table 1) there was a significant interaction between strain and diet ($F = 27.2$; $df = 3, 36$; $P < 0.001$). On both diets the laboratory mites had a shorter pre-reproductive period than the wild mites. Wild mites had a significantly longer pre-reproductive period on the skin/dust diet compared to the liver/yeast diet ($F = 32.0$; $df = 1, 18$; $P < 0.001$), but no effect of diet was seen in laboratory mites.

At 64% RH (Table 2) there were no significant differences between strains, and in both strains the skin/dust diet resulted in significantly longer pre-reproductive periods compared to the liver/yeast diet ($F = 18.9$; $df = 1, 18$; $P < 0.001$).

Reproductive Period.

The three-way interaction between strain, diet and RH was significant ($F = 33.1$; $df = 7, 72$; $P = 0.008$). On the liver/yeast diet both strains of mites showed markedly shorter reproductive periods at 64% RH compared to 75% ($F = 68.4$; $df = 2, 36$; $P < 0.001$). There was no significant response to RH in wild mites on the skin/dust diet, but in the lab strain fed on the skin/dust diet reproductive period was significantly longer at 64% than at 75% RH ($F = 5.9$; $df = 1, 18$; $P = 0.026$) (Tables 1 and 2).

There was no influence of diet on reproductive period at 75% RH and the only significant difference between strains was seen on the liver/yeast diet where wild

mites had a longer mean reproductive period than laboratory mites ($F = 68.4$; $df = 2, 36$; $P < 0.001$) (Table 1).

At 64% RH (Table 2) the magnitude of the response to diet differed between the two strains of mites ($F = 35.8$; $df = 3, 36$; $P = 0.003$). While both strains of mites showed significantly shorter reproductive periods on the liver/yeast diet compared to the skin/dust diet, the response was greater in the laboratory strain. As found at 75% RH, wild mites had a longer mean reproductive period than laboratory mites on the liver/yeast diet. In contrast, on the skin/dust diet laboratory mites had a significantly longer reproductive period than wild mites.

Reproductive Rate.

Reproductive rate was higher at 75% RH than at 64% in both mite strains and higher in the laboratory strain than in the wild strain at both RH levels ($F = 27.0$; $df = 2, 77$; $P < 0.001$) (Tables 1 and 2). Diet did not affect reproductive rate.

Female Survival.

Female survival (the period from mating of female tritonymphs with males and death) of both mite strains decreased at 64% RH compared to 75% RH on the liver/yeast diet ($F = 57.8$; $df = 3, 36$; $P < 0.001$) and this decline in survival was more marked in the laboratory mite strain than in the wild strain. On the skin/dust diet female survival increased with RH in the wild strain, but decreased with increasing RH in the laboratory strain ($F = 5.0$; $df = 3, 37$; $P = 0.005$) (Tables 1 and 2).

Diet had no significant effect on female survival at 75% RH and the only difference between strains at this RH was found on the liver/yeast diet, where survival of wild females was greater than laboratory-reared females ($F = 57.8$; $df = 3, 36$; $P < 0.001$) (Table 1).

At 64% RH (Table 2) the two mite strains differed in the magnitude of their response to diet ($F = 71.9$; $df = 3, 36$; $p < 0.001$). While female survival of both mite strains was lower on the liver/yeast diet compared to the skin/dust diet, the laboratory strain demonstrated a much greater increase in survival on the skin/dust

diet than did the wild mites. Wild females on the liver/yeast diet had greater survival than laboratory mites on this diet, whereas the laboratory strain had much greater survival than the wild strain on the skin/dust diet.

Male Survival.

The three-way interaction between mite strain, diet and RH was a significant predictor of male survival ($F = 20.8$; $df = 7, 71$; $P < 0.001$). Survival of males (survival time of males of unknown age during experiments) of both mite strains decreased at 64% RH compared to 75% RH on the liver/yeast diet ($F = 125.3$; $df = 1, 37$; $P < 0.001$). On the skin/dust diet this effect was seen only in the wild mite strain ($F = 29.5$; $df = 3, 35$; $P < 0.001$) and was less marked than that seen on the liver/yeast diet (Tables 1 and 2).

At 75% RH (Table 1) there was no effect of mite strain on male survival, however wild males on the skin/dust diet had greater survival than those on the liver/yeast diet at this RH ($F = 29.5$; $df = 3, 35$; $P < 0.001$).

At 64% RH (Table 2) again diet was the only significant predictor of male survival, with males of both strains on the skin/dust diet living longer than those on the liver/yeast diet ($F = 125.3$; $df = 1, 37$; $P < 0.001$).

Immature Development.

Only the first principal component had an eigenvalue greater than one, and it explained 78.8% of the variation in egg, juvenile and total development. PC1 was positively correlated with all of these factors, and had high factor loadings of all, suggesting that the dependent variables are highly inter-correlated and likely to show similar patterns among the ANOVAs.

The significant interactions between strain/RH and strain/diet in predicting PC1 ($F = 85.6$; $df = 5, 255$; $P < 0.001$) show that strain, diet and RH are all significant predictors of immature development. This demonstrates that effects detected within each developmental trait were real, and not artefacts of accepting random patterns as significant due to the number of separate tests done.

Egg Development.

The three-way interaction between mite strain, diet and RH was a significant predictor of egg development times ($F = 91.1$; $df = 7, 333$; $P < 0.001$). Compared to 75% RH, egg mortality at 64% was 40% higher in laboratory mites on the liver/yeast diet. On this diet eggs of both strains of mites developed faster at 75% RH compared to 64% ($F = 111.6$; $df = 7, 333$; $P < 0.001$). However, on the skin/dust diet, while the laboratory strain showed more rapid egg development at 75% compared to 64%, egg development in the wild strain was inhibited at 75% RH ($F = 100.6$; $df = 7, 333$; $P < 0.001$) (Tables 1 and 2).

At 75% RH (Table 1) there was no effect of diet on egg development, but eggs from the laboratory strain developed more quickly than those laid by the wild strain on both diets ($F = 52.7$; $df = 1, 198$; $P < 0.001$).

At 64% RH both mite strain and diet influenced egg development (Table 2). Development times were quicker when the laboratory mite strain was on its accustomed diet (liver/yeast) compared to the skin/dust diet and also when the wild mites were on their accustomed diet (skin/dust) compared to the liver/yeast diet ($F = 194.3$; $df = 1, 198$; $P < 0.001$). Strain effects were seen on the liver/yeast diet where wild mite eggs had much slower development than eggs from the laboratory strain ($F = 111.6$; $df = 7, 333$; $P < 0.001$), whilst the reverse was true on the skin/dust diet.

Juvenile Development.

No juveniles of either strain of mites completed development on the liver/yeast diet at 64% RH, thus comparisons could be made between strain and RH on the skin/dust diet only. At 75% RH both strains had faster juvenile development than at 64% ($F = 70.4$; $df = 5, 294$; $P < 0.001$) (Tables 1 and 2).

At 75% RH, juvenile development responded differently to diet between strains ($F = 24.3$; $df = 3, 196$; $P < 0.001$). Each strain had faster development on the diet to which they were accustomed compared to the alternative diet (Table 1).

At 64% RH, on the skin/dust diet the wild mites had slower juvenile development than the laboratory strain on this diet ($F = 60.6$; $df = 3, 196$; $P < 0.001$) (Table 2).

Total Development.

There was a significant three-way interaction of mite strain, diet and RH in predicting total development time ($F = 72.3$; $df = 4, 295$; $P < 0.001$). Total development was faster at 75% RH than at 64% RH in both strains (Tables 1 and 2).

At 75% RH, in the laboratory mite strain the skin/dust diet markedly delayed total development compared to the liver/yeast diet, but no significant effect of diet was detected in the wild strain ($F = 12.0$; $df = 1, 198$; $P < 0.001$) (Table 1).

At 64% RH there were no significant differences in total development of the two mite strains on the skin/dust diet (Table 2).

Discussion

This study has provided life history parameters of laboratory-reared *D. pteronyssinus* on a laboratory diet at 25°C and 75% RH, which generally appear to have higher reproductive parameters and faster development than previous reports (Spieksma 1967, Blythe 1976, Dobson 1979, Gamal-Eddin *et al.* 1983a, 1983b, 1983c; Ho and Nadchatram 1984, Colloff 1987a, 1987b; Andersen, 1988, Hart and Fain 1988, Arlian *et al.* 1990). Differences between these results are likely to be due to differences in strain of mites and/or diet. We have demonstrated the importance of diet in this paper and we are also currently investigating the extent to which different strains of wild mites may vary in their life history parameters.

There have been few published studies on life history parameters of laboratory-reared *D. pteronyssinus* at 25°C and 64%RH. However, our laboratory mites appeared to perform less well at low RH than previous reports (Gamal-Eddin *et al.* 1983a, 1983b, 1983c; Colloff 1987a, 1987b). This may be due to the liver/yeast diet of desiccated liver and yeast, which appeared to be unsuitable for mite reproduction and development at low RH compared to the skin/dust diet (see discussion below).

This study has also provided the first comprehensive data set of adult reproduction and immature survival and development of wild *D. pteronyssinus* in optimal and sub-optimal rearing conditions. Previously only egg survival and development has been reported by Colloff (1987a, 1987b), who suggested that eggs of wild mites survive better and develop more quickly than those of laboratory mites when reared in cool, dry conditions of temperature and RH, whereas in warm, humid conditions the reverse is true. Our results also suggest that laboratory mites perform better (higher fecundity and rate of reproduction, shorter pre-reproductive period and faster egg development) than wild mites in optimum rearing conditions (75% RH), but in sub-optimum conditions (64% RH) laboratory-reared mites perform less well (lower fecundity, shorter reproductive period, reduced female survival and higher egg mortality) on the liver/yeast diet than wild mites. In contrast, on the skin/dust diet at low RH, fecundity, rate of reproduction, reproductive period and female survival of laboratory mites were higher than found in wild mites, but rearing the laboratory mites prior to the experiment on an optimised liver/yeast diet is likely to have had an influence on their subsequent egg production and survival on the skin/dust diet.

In some arthropods specific traits can be selected after as little as five generations (Navarro *et al.* 1985, Yano and Takafuji 2002, Young *et al.* 2003). Therefore, during the period between collection and the start of experiments, it is possible that our wild mite cultures may have in part adapted to laboratory culture conditions and thus represent an intermediate stage between wild and fully adapted long-term laboratory cultures. However this seems unlikely, since our wild cultures were reared in conditions relating very closely to those found in the home (diurnally fluctuating hygrothermal conditions) and on a natural diet consisting of only skin scales and house dust.

The poor performance of adults and immatures on the liver/yeast diet compared to the skin/dust diet at low RH was particularly striking. Most existing data on reproduction and development of house dust mites have been obtained from mites reared on laboratory diets which are highly nutritious and provide good population development in optimum hygrothermal conditions, but such diets may not provide an ideal substrate for mite survival at low RH. This could explain the sparse data on survival, reproduction and development of house dust mites reared

at RH below 75%. However, Saint Georges-Gridelet (1984) previously reported high population growth of *D. pteronyssinus* at 64% RH on diets high in lipids. In this paper it was suggested that lipids present in skin scales in the house dust substrate could explain the survival of mites in their natural habitat where hygrometric conditions are often below the critical equilibrium activity (Arlian 1975, Arlian and Veselica 1981) of the mites. Our results appear to agree with this and suggest that data on mite performance on laboratory yeast-based diets, particularly in sub-optimal conditions, are unlikely to represent performance on a skin-based diet in their natural dust habitat.

Population models to predict dust mite populations in homes are currently under development using previously published data primarily from laboratory populations of mites reared on laboratory diets (Pretlove *et al.* 2001, 2005; Crowther *et al.* 2006, Biddulph *et al.* 2007). The present study has highlighted the requirement for a more comprehensive data set from wild mite populations reared on a natural diet for use in these models. Work is underway by the current authors to provide this data.

Another critical factor likely to have an influence on the life history parameters of house dust mites is fluctuating temperature and RH. De Boer *et al.* (1998) have shown that *D. pteronyssinus* can survive and produce eggs when held at low RH and given as little as 3 hours moist air per day. Arlian *et al.* (1999) suggested that the development of *D. farinae* (Hughes) was slower in fluctuating conditions of RH compared to constant high RH. More recently Pike *et al.* (2005) found that the population dynamics for *D. pteronyssinus* were similar in both fluctuating and constant conditions of temperature and RH. However, Colloff (1987a) proposed that laboratory mite populations were less able to withstand diurnal fluctuations in microclimate than wild populations of mites. This is also the subject of a future paper by the current authors using wild mites reared on a natural diet.

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References Cited

Andersen, A. 1988. Population growth and developmental stages of the house dust mite, *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). J. Med. Entomol. 25: 370-373.

Arlian, L.G. 1975. Dehydration and survival of the European house dust mite, *Dermatophagoides pteronyssinus*. J. Med. Entomol. 12: 437-442.

Arlian, L.G., J.S. Neal, and D. Vyszenski-Moher. 1999. Fluctuating hydrating and dehydrating relative humidities effects on the life cycle of *Dermatophagoides farinae* (Acari: Pyroglyphidae). J. Med. Entomol. 36: 457-461.

Arlian, L.G., C.M. Rapp, and S.G. Ahmed. 1990. Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). J. Med. Entomol. 27: 1035-1040.

Arlian, L.G., and M.M. Veselica. 1981. Re-evaluation of the humidity requirements of the house dust mite *Dermatophagoides farinae* (Acari: Pyroglyphidae). J. Med. Entomol. 18: 351-352.

Biddulph, P., D. Crowther, B. Leung, T. Wilkinson, B. Hart, T. Oreszczyn, S. Pretlove, I. Ridley, and M. Ucci. 2007. Predicting the population dynamics of the house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle. Exp. Appl. Acarol. (in press).

Blythe, M.E. 1976. Some aspects of the ecological study of the house dust mite. Brit. J. Dis. Chest 70: 3-31.

Boer, R., K. de Kuller, and O. Kahl. 1998. Water balance of *Dermatophagoides pteronyssinus* (Acari : Pyroglyphidae) maintained by brief daily spells of elevated air humidity. J. Med. Entomol. 35: 905-910.

Colloff, M.J. 1987a. Differences in development time, mortality and water loss between eggs from laboratory and wild populations of *Dermatophagoides pteronyssinus*. Exp. Appl. Acarol. 3: 191-200.

Colloff, M.J. 1987b. Effects of temperature and relative humidity on development times and mortality of eggs from laboratory and wild populations of the European house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Exp. Appl. Acarol. 3: 279-289.

Crowther, D., T. Wilkinson, P. Biddulph, T. Oreszczyn, S. Pretlove, and I. Ridley. 2006. A

simple model for predicting the effect of hygrothermal conditions on populations of house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Exp. Appl. Acarol. 79: 127-148.

Cunningham, M.J. 1999. Modelling of some dwelling internal microclimates. Building and Environment 34: 523-536.

Dobson, R.M. 1979. Some effects of microclimate on the survival and development of *Dermatophagoides pteronyssinus* (Trouessart). Acarologia 2: 482-486.

Gamal-Eddin, F. M., F.M. Abou-Sinna, S.E. Tayel, A.M. Aboul-Atta, A.M. Seif. and S.M. Gaafar. 1983a. Duration of the developmental stages of house dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled temperatures and relative humidities to pave the way in front of the workers in the field of house dust mite asthmatic bronchitis. 1. Pre-imaginal period. J. Egypt. Soc. Parasitol. 13: 319-334.

Gamal-Eddin, F.M., K.K. Shehata, F. Abou-Sinna, S. Tayel, A.M. Aboul-Atta, B.A. El-Ahmedaawy, M.A. Fayed, A.H. Hafez, and M. Imam. 1983b. Duration of the development stages of house-dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled conditions to pave the way in front of the workers in the field of house-dust mite asthmatic bronchitis. 2. Oviposition period, fecundity and oval duration. J. Egypt. Soc. Parasitol. 13: 557-581.

Gamal-Eddin, F.M., K.K. Shehata, F.M. Abou-Sinna, S.E. Tayel, A.M. Aboul-Atta, B.A. El-Ahmedaawy, M.A. Fayed, A.H. Hafez, and M. Imam, M. 1983c. Duration of the developmental stages of house dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled conditions to pave the way in front of the workers in the field of house dust mite asthmatic bronchitis 3. Larval Duration. J. Egypt. Soc. of Parasitol. 13: 583–595.

Hart, B.J., and A. Fain. 1988. Morphological and biological studies of medically important house-dust mites. Acarologia 29: 285-295.

Ho, T.M., and M. Nadchatram. 1984. Life cycle and survival of *Dermatophagoides pteronyssinus* (Trouessart) (Acarina: Astigmata: Pyroglyphidae) in a tropical laboratory. Trop. Biomed. 1: 159-162.

Legendre, P. and L. Legendre. 1998. Numerical Ecology. 2nd ed. Elsevier Publ, The Netherlands.

Navarro, S., R. Dias, and E. Donahaye. 1985. Induced tolerance of *Sitophilus oryzae* adults to carbon dioxide. J. Stored Products Res. 21: 207-213.

Pike, A.J., M.J. Cunningham, and P.J. Lester. 2005. Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) at constant and simultaneously fluctuating temperature and humidity conditions. J. Med. Entomol. 42: 266-269.

Pretlove, S., I. Ridley, J. Horwood, B. Leung, P. Cox, D. Thomson, N. Baker, D. Crowther, and T. Oreszczyn. 2001. A combined transient hygrothermal and population model of house dust mites in beds. Proceedings of IAQ 2001: Moisture, Microbes and Health Effects: Indoor Air Quality and Moisture in Buildings, ASHRAE, San Francisco, USA, November 4 – 7, 2001.

Pretlove, S., R. Critchley, R. Howard, and T. Oreszczyn. 2002. The Nottingham energy, health and housing study: reducing relative humidity, dust mites and asthma. Building Services Engineering Research & Technology 23: 43-55.

Pretlove, S., T. Oreszczyn, I. Ridley, T. Wilkinson, and D. Crowther. 2005. A steady state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements. Building Services Engineering Research & Technology 26 (4): 301-314.

Saint Georges-Gridelet, D. de 1984. Effects of dietary lipids on the population growth of *Dermatophagoides pteronyssinus*. Acarology 6: 351-357.

Sokal, R.R., and F.J. Rohlf. 1995. Biometry. WH Freeman Publ., USA.

Spieksma, F.Th.M. 1967. The House Dust Mite *Dermatophagoides pteronyssinus* (Trouessart, 1897). PhD Thesis, Univ. of Leiden, 65 pp.

Winston, P.W., and D.H. Bates. 1960. Saturated solutions for the control of humidity in biological research. Ecology 41: 232-237.

Yano, S., and A. Takafuji. 2002. Variation in the life history pattern on *Tetranychus urticae* (Acari: Tetranychidae) after selection for dispersal. Exp. Appl. Acarol. 27: 1-10.

Young, H.P., D.W. Bailey, and R.M.R.M. Roe. 2003. Spinosad selection of a laboratory strain of the tobacco budworm, *Heliothis virescens* (Lepidoptera: Noctuidae), and characterization of resistance. Crop Protection 22: 265-273.

Table 1. Life history parameters of laboratory and wild populations of *D. pteronyssinus* (DP) reared on liver/yeast (lab) and skin/dust (dust) diets at 25ūC and 75% RH.

	25ūC, 75% RH			
	Lab DP Lab diet	Wild DP Lab diet	Lab DP Dust diet	Wild DP Dust diet
ADULTS (n=10)				
Total fecundity per female	100 ± 33.4	66.9 ± 15.9	79.7 ± 12.6	60.9 ± 15.9
Pre-reproductive period (days)	2.2 ± 0.6	2.9 ± 1.1	2.6 ± 0.9	8.3 ± 3.4
Reproductive period (days)	35.5 ± 12.9	43.1 ± 22.9	27.0 ± 8.2	35.9 ± 18.9
Rate of reproduction (eggs/female/day)	3.0 ± 1.1	1.7 ± 0.5	3.2 ± 1.0	1.9 ± 0.8
Female survival (days)	45.2 ± 13.8	52.8 ± 22.1	39.9 ± 17.5	48.6 ± 20.2
Male survival (days)	34.8 ± 14.4	31.2 ± 5.9	33.9 ± 21.1	45.4 ± 16.1
IMMATURES (N=50)				
% egg mortality	0	0	0	0
Egg development time (days)	3.5 ± 0.9	4.5 ± 0.8	3.3 ± 0.5	5.4 ± 2.8
% juvenile mortality	0	0	0	0
Juvenile development time (days)	9.8 ± 1.2	10.9 ± 1.7	13.1 ± 0.7	10.1 ± 3.5
Total egg-adult development time (days)	13.3 ± 1.4	15.4 ± 2.4	16.4 ± 0.7	15.4 ± 4.4

Results show Mean ± SD.

Table 2. Life history parameters of laboratory and wild populations of *D. pteronyssinus* (DP) reared on liver/yeast (lab) and skin/dust (dust) diets at 25ūC and 64% RH.

	25ūC, 64% RH			
	Lab DP Lab diet	Wild DP Lab diet	Lab DP Dust diet	Wild DP Dust diet
ADULTS (n=10)				
Total fecundity per female	3.8 ± 0.9	6.9 ± 3.2	48.0 ± 10.3	24.4 ± 5.5
Pre-reproductive period (days)	6.2 ± 1.9	6.0 ± 1.6	12.4 ± 6.2	10.1 ± 3.7
Reproductive period (days)	2.8 ± 1.9	6.3 ± 3.5	36.2 ± 8.8	29.4 ± 10.3
Rate of reproduction (eggs/female/day)	2.3 ± 1.7	1.3 ± 0.7	1.4 ± 0.6	0.8 ± 0.4
Female survival (days)	10.2 ± 2.4	18.7 ± 4.8	56.6 ± 13.1	33.1 ± 7.8
Male survival (days)	9.8 ± 2.9	11.6 ± 4.0	44.0 ± 14.1	37.9 ± 12.8
IMMATURES (N=50)				
% egg mortality	40	0	0	0
Egg development time (days)	4.5 ± 1.1	8.0 ± 0	8.0 ± 0	2.3 ± 1.0
% juvenile mortality	100	100	0	0
Juvenile development time (days)			16.0 ± 3.5	23.9 ± 9.8
Total egg-adult development time (days)			24.0 ± 3.5	26.0 ± 9.9

Results show Mean ± SD.

Appendix A.0: Published Papers

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A.0.4: *The psychrometric control of house dust mites: a pilot study*

M Ucci[a], SEC Pretlove[b], P Biddulph[c], T Oreszczyn[a], T Wilkinson[c,d] D Crowther[c] G Scadding[e], B. Hart[f], D Mumovic[a]

[a]The Bartlett School of Graduate Studies, University College London, London, UK; [b]Kingston University, School of Architecture & Landscape, London, UK; [c]The Martin Centre, Department of Architecture, University of Cambridge, Cambridge, UK; [d]The Medical Entomology Centre, Shepreth, Royston, UK; [e]Royal National Throat Nose and Ear Hospital, London, UK; [f]The Royal Agricultural College, Cirencester, UK.

Abstract

This paper describes a pilot intervention study on the effectiveness of house dust mite allergen avoidance for twelve asthmatic children (two being controls). In addition to mite allergen removal, the study included tailored advice aimed at reducing mite population growth via changes in moisture production, heating and ventilation habits. This paper focuses on the effects of this advice on household behaviour, hygrothermal conditions and mite populations. The efficacy of monitoring and modelling techniques is also discussed. The study highlighted a number of interrelated confounding factors which have to be addressed in future similar larger scale studies, but the results are promising with regards to the effectiveness of such studies.

Practical application

This study suggests that in temperate climates tailored advice on moisture production, heating and ventilation habits can lead to valuable changes in hygrothermal conditions, which in turn can result in reduced mite populations. However, pre-existing adverse building conditions may hinder such changes, and the effectiveness of tailored advice and of hygrothermal modifications is often difficult to assess. It is therefore recommended that any similar larger intervention study measures ventilation rates and adequately controls for a number of confounding variables - including the effect of changes in outdoor conditions and of the removal of existing mite populations. In this respect, hygrothermal population models can play a very useful role in the assessment of study effectiveness.

1.0 Introduction

House dust mites (HDM) can be found in beds, carpets and soft furnishings. Exposure to them can lead to allergic sensitisation and to rhinitis and asthma

symptoms. Noticeable differences have been found in the prevalences of atopy and asthma symptoms worldwide, with the UK having some of the highest values[1].

House dust mites absorb moisture from the air. If the ambient RH is too low, mites dehydrate and eventually die. This critical low RH is often referred to as the *Critical Equilibrium Humidity* (CEH), which is probably temperature-dependent for *Dermatophagoides pteronyssinus* (DP), the most common species in the UK[2]. Temperature also plays an important independent role in mite physiology, for example affecting egg-to-adult development times. Thus, by adequately controlling the hygrothermal conditions of mite microclimates (psychrometric control)[3], it should be possible to reduce mite populations. Because of the dependency of mite populations on hygrothermal conditions, their growth in temperate climates is usually greatest in late summer/early autumn, and least in the winter months, which are a crucial time for reducing mite populations[2]. For typical indoor temperatures, maintaining the average daily indoor RH below 50% is often recommended to reduce mite levels and their allergens. HDM can survive when exposed to brief spells of high RH, even when the daily average RH is below critical levels[4,5]. Nonetheless, the psychrometric control method is still viable, as mite development rates are much slower under such circumstances[4].

Most studies on the psychrometric control of house dust mites in housing have focused on mechanical ventilation. There is however scope for modifying residential hygrothermal conditions by changing heating and ventilation habits. For example, a UK study found that extractor fans in the kitchen were associated with lower HDM allergen concentrations[6]. Nonetheless, few intervention studies have attempted to reduce HDM levels through modifications of occupant behaviour alone. In winter 2005 the authors took part in a pilot study on the effectiveness of dust mite allergen avoidance for 12 asthmatic children. It was filmed by *Twenty Twenty Television*[7] and resulted in two 50-minutes episodes of the UK TV series 'Dispatches' on Channel 4 (April 2006). Due to its short time-scale and small sample size, the study did not aim to establish the clinical efficacy

of allergen avoidance, but to illustrate potential benefits and give researchers the opportunity to test a protocol for a larger future study. As well as the removal of mite and pet allergens, it included tailored advice aimed at reducing mite population growth via changes in moisture production, heating and ventilation habits. The study addressed four issues: 1) the effect of allergen removal on the children's health; 2) the effect of tailored advice on occupant behaviour and the resultant hygrothermal conditions; 3) the effect of the hygrothermal changes on mite populations; and 4) the efficacy of monitoring/modelling techniques. This paper will focus on the last three issues, however the results did reveal a (weak) correlation between health improvements and HDM allergen reduction. This is encouraging, particularly considering the limited statistical power of this study. This paper illustrates the methods and findings of the pilot study, with a view to discussing their implications for a future larger scale study.

2.0 Methodology

In October 2005, twelve asthmatic mite-sensitive children aged 6 to14 were selected in the London area by the TV production team[7]. Eleven dwellings were examined overall, since two of the children were siblings living in 1 dwelling (here termed bedroom/child 12a and 12b). The properties included: 4 flats, 1 detached house and 6 terraced houses. A pre-intervention analysis was carried out in November 2005, where baseline measurements were taken of: the children's health status; HDM numbers and allergen levels in each dwelling (child bedroom: mattress, pillow, one soft toy and floor; living room: sofa and floor - all using a standard protocol); hygrothermal conditions (monitored for 2 weeks, logging every 15 minutes); building characteristics (including airtightness via a fan-pressurisation test); and heating and ventilation habits. The fan-pressurisation results at 50 Pa were converted to an estimated air-infiltration rate in air changes per hour under average external conditions[8]. The children's asthma and health status was assessed by Dr Glenis Scadding, consultant physician at the Royal National Throat Nose & Ear Hospital. After the pre-intervention study, a number of interventions were carried out, followed by a post-intervention study, where the

children's health and the dwellings' hygrothermal conditions were monitored for 6 weeks (Dec 05-Jan 06).

After the baseline measurements, the following interventions were carried out: professional steam-cleaning of the child's bedroom and thorough cleaning of the dwelling (followed by further dust sampling); replacement of carpets in the child's bedroom with laminate flooring; covering mattresses, pillows and duvets with micro-porous mite-proof barriers; removing pets and cuddly toys; and avoiding exposure to environmental tobacco smoke. The participants were also advised to implement a thorough cleaning regime throughout the post-intervention period. Following the analysis of the pre-intervention study results, tailored advice was also provided on moisture production, heating and ventilation. Outdoor hygrothermal conditions were also monitored throughout the study. For the two households acting as controls, the interventions were carried out *at the end* of the post-intervention period, but their dwelling's hygrothermal conditions were monitored throughout the study. At the end of the study, further dust samples were taken, and a final medical examination was carried out.

3.0 Pre Intervention Study: Baseline Measurements and Hygrothermal Advice

Table 1 shows the baseline results. Since little dust was found in toys and pillows, it was concluded that allergen concentrations can be misleading at times. Therefore, the results were also expressed in terms of 'allergen load': total allergen weight collected for a given vacuumed area, corresponding to μg Der p1/m^2 (μg Der p1/total object area, for pillows and toys). The daily moisture production (kg/day) was estimated by using the questionnaire results and the moisture algorithm of Condensation Targeter II[9]. The Critical Equilibrium Humidity (CEH) was calculated as a function of temperature using DF data, which is the fullest dataset currently available[10].

Based on the pre-intervention study results illustrated in Table 1, tailored advice was provided to each household on the most appropriate heating, ventilation and moisture-production patterns, which could reduce house dust mite populations. Depending on the dwelling and occupant behaviour characteristics, each household was advised to implement one, or a combination of, the following measures: a) reducing moisture production; b) increasing ventilation levels; and c) increasing temperature levels.

For example, Household 1 – with low air infiltration rates, and highest RH and VPX levels – was advised to: 1) only dry clothes indoors in a well ventilated room, which is closed to the rest of the home; 2) use the extract fans in the kitchen and the bathroom during use, and for at least 15 minutes afterwards; 3) keep the trickle vents always open; and 4) leave the windows slightly open, for as long as possible. On the other hand, Household 11 - with low temperatures and high infiltration rates - was advised to increase indoor temperatures. Control households (6 and 8) did not receive the advice until the end of the study.

4.0 Post-intervention results

In all dwellings, the bedroom RH decreased from pre to post intervention periods, and the percentage of time the bedroom RH was greater than CEH ('% time RH>CEH') also decreased (Table 2). However, the reduction in indoor RH levels was partly due to changes in outdoor conditions. Although the average outdoor RH increased during the post-intervention, it was colder and the outdoor absolute humidity was lower. In order to disentangle the weather effect from the advice implementation, the pre and post intervention RHs were adjusted for each bedroom. It was assumed that if the pre and post intervention period had exactly the same weather conditions, the indoor temperatures would be the same, and the indoor RH would be dependent on the dwelling's vapour pressure excess, as well as on the outdoor vapour pressure. Since the outdoor conditions had been monitored for a longer period during the post intervention period, the indoor RHs were adjusted in relation to the post intervention weather conditions. The adjusted vapour pressure was calculated as follows:

$$Pre\_VP^{Adj} = Out\_VP + Pre\_VPX \quad [1]$$

$$Post\_VP^{Adj.} = Out\_VP + Post\_VPX \quad [2]$$

where Out_VP is the outdoor vapour pressure monitored during the post intervention study; Pre_VPX is the average vapour pressure excess measured during the pre intervention period; Post_VPX is the average vapour pressure excess measured during the post intervention period. Figure 1 illustrates a schematic representation of the adjustment calculation for the vapour pressure. The adjusted RH was then calculated as follows:

$$Pre\_RH^{Adj} = RH(Post\_T, Pre\_VP^{Adj}) \quad [3]$$

$$Post\_RH^{Adj} = RH(Post\_T, Post\_VP^{Adj}) \quad [4]$$

where Pre_RH^{Adj} is the adjusted pre intervention RH, as a function of the monitored post temperature (Post_T), and the adjusted pre vapour pressure (Pre_VP^{Adj}). The $Post\_RH^{Adj}$ is the adjusted post intervention RH, as a function of the monitored post temperature (Post_T), and the adjusted post vapour pressure ($Post\_VP^{Adj}$).

Once the impact of changes in outdoor conditions was taken into account, it was found that the reduction in bedroom RHs was smaller than the measured results (Figure 2) - particularly for some bedrooms (measured pre-post average RH difference: 12.1%; adjusted pre-post average RH difference: 5.1%, excluding control bedrooms). A paired t-test showed that there was a statistically significant difference ($p<0.01$) between pre and post bedroom RHs, for both the *measured* and the *adjusted* RH results. The importance of the adjustment procedure is highlighted by the case of the control Dwelling 8, where the measured bedroom RH decreased from the pre to the post intervention period. However, its adjusted RH *increased* (by a small amount, Fig 2). This was due to an increase in the measured post-intervention vapour pressure excess (Table 2), probably because of a reduction in window opening for the colder outdoor temperatures. It should also be highlighted that the other control dwelling (Dwelling 6) experienced an above average reduction in adjusted RH levels. However it is possible that Household 6 learnt about the advice provided to other families. It is also possible that the RH adjustment method described earlier may underestimate reductions in relative

humidity, since the VPX in dwellings can be dependent on outdoor conditions, with higher VPXs during cold weather - probably because occupants ventilate less when it is cold outside[11]. Furthermore, the adjustment method is more suitable for identifying changes in RH due to reduction in VPX, rather than to changes in temperatures.

At the end of the intervention study, tailored interviews were carried out in order to establish further the extent to which households had implemented the advice. Based on the interview results, each household was given an 'implementation score'. No correlation was found between the measured pre/post RH reduction and the 'implementation score'. Therefore, for those dwellings which experienced small reductions in RH, it is difficult to establish whether this was due to: a) lack of participants' action, b) adverse building characteristics hindering changes, c) limitations of the advice itself. However, during the interviews it also became apparent that participants experienced some difficulties in reporting their ventilation habits coherently.

5.0 The role of modelling techniques

The population model Mite Population Index (MPI)[2] was utilised in this study in order to: 1) Help identify those dwellings most at risk from mite growth; 2) Assess the effect of changes in hygrothermal conditions on mite populations. The MPI model predicts the likely effect of steady-state average hygrothermal conditions, on HDM population growth. The output is the MPI index, where for example 1.1 indicates 10% population growth and 0.9 indicates 10% population decline. The results (Table 3) obtained by utilizing measured pre-intervention average conditions indicate that the mite populations were rather stable during that period (average MPI $\cong$ 1), suggesting that even small hygrothermal changes could determine whether the population grows or declines (threshold effect). Dwelling 1 had the highest predicted daily population growth for both bedroom and bed. This is in agreement with the results from the dust samples. The other bedrooms with high MPI values did not have the highest allergen levels. This is

probably due to a reservoir effect, where for example the age of the mattress or cleaning regimes affect allergen levels.

Since the interventions included the removal of mites and their allergens, it was not possible to assess the *direct* effect of RH reductions on mite levels in the dwelling. Modelling was therefore utilised in order to assess the likely impact of hygrothermal changes on mite populations. In order to exclude the effect of changes in outdoor conditions, the monitored post-intervention temperatures and the *adjusted* post-intervention bedroom RHs were used as inputs in the MPI model. The use of adjusted hygrothermal conditions is theoretically equivalent to a situation where outdoor conditions stayed the same throughout the whole study, which allows the assessment of the likely impact of advice implementation on mite populations. Figure 3 shows a plot of the adjusted average hygrothermal conditions, with the pre and the post conditions joined by an arrow for each bedroom (numbers near data points correspond to the bedrooms' code). The plot includes a curve – corresponding to an MPI value of 1 – above which mite populations grow, and below which they decline. The results show that in all but Dwelling 8, conditions improved – independently from weather changes. Because of the threshold effect, even small reductions in RHs can lead to a reduction in mite populations (e.g. Bedroom 1 in Fig 3). Therefore, although the RH reductions obtained via changes in occupant behaviour may appear small, they could be sufficient in reducing mite infestations – particularly in winter times.

6.0 Discussion

This paper illustrated the methods and the findings of a pilot intervention study on HDM allergen avoidance, with a view to discussing their implications for a future larger scale study. The paper focused on the effects of tailored advice for heating and ventilation habits on household behaviour, indoor hygrothermal conditions and mite populations, as well as the role of modelling techniques. The results highlight the complexities associated with these types of studies, which are affected by several confounding factors[12]. Firstly, the study shows that, depending

on the time of the year, changes in outdoor conditions may result in improved hygrothermal conditions, thus confounding the effect of hygrothermal changes due to the advice implementation. Furthermore, occupant behaviour can change in relation to outdoor conditions. It is therefore important to adjust for changes in outdoor conditions in future studies.

Secondly, the study shows that changes in occupant behaviour can be difficult to assess – particularly with regard to ventilation habits. It is therefore vital that ventilation rates – not only air leakage – are measured in future studies, and a closer monitoring is carried out of occupant habits (for example through the use of participants diaries). Future studies might also need to focus on similar building types, in order to facilitate the assessment of changes in occupant behaviour. As demonstrated in this study, it is vital that *tailored* advice is provided, ensuring that the most effective intervention is carried out - since greater ventilation rates are not always desirable (e.g. leaky building). Changes in occupant behaviours can also be hindered by adverse dwelling characteristics. For example, in this study Household 1 was advised to increase ventilation rates, but their VPX did not decrease, most probably because of a combination of existing habits and their very airtight dwelling.

A further difficulty highlighted by this study was assessing the impact of changes in hygrothermal conditions on mite populations (and, indirectly, on health). This is because psychrometric control does not immediately affect HDM allergen reservoirs, which have to be removed in order to obtain any health improvement. However, allergen removal also results in killing the existing mite population, which therefore cannot be monitored for subsequent reductions due to hygrothermal changes. In any case, live mites are notoriously difficult to sample and mite populations would also be affected by changes in outdoor conditions. On the other hand, high allergen levels cannot be taken as a marker of favourable hygrothermal conditions, since a reservoir effect can be observed, for example due to the age of the substrate (e.g. mattress) and to cleaning regimes. Therefore, in future studies the use of population modelling techniques such as those utilised in

this pilot are recommended as a very useful tool for assessing the likely impact of hygrothermal changes on mite populations. Although the current models are based on steady-state data, transient models for mite microclimates are being developed by the authors, which will allow for more accurate predictions of mite populations. Furthermore, the authors have also developed an innovative technique for monitoring the impact of a dwelling's hygrothermal conditions on mite populations - described in a future paper. The development of models predicting the effect of hygrothermal conditions on allergen levels (as opposed to mite population only) is also recommended, since this would allow for a better understanding of the impacts of hygrothermal changes on respiratory health.

Due to practical constraints, the placebo effect and the role of other confounding health variables (e.g. sensitivity to other allergens) could not be adequately controlled for in this study and they should be properly addressed at the design stage in future studies (e.g. eliminating children who are allergic to other allergens). In this pilot one control dwelling achieved a large reduction in bedroom RHs – despite not receiving the advice until after the end of the study. This may have been due to the nature of this pilot (role of TV crew) but larger future studies should attempt to control for this by ensuring minimum interaction between participants, and with the project team.

This study showed that there was a statistically significant ($p<0.01$) decrease of measured bedroom RHs, which remained statistically significant although smaller, once the effect of changes in outdoor conditions was taken into account. The population modeling results indicated that during the pre-intervention period the mite populations were rather stable (average MPI $\cong$ 1), suggesting that even *small* hygrothermal changes could determine whether mite populations grow or decline.

7.0 Conclusions

This study suggests that in temperate climates even small changes in hygrothermal conditions can be crucial for the reduction of house dust mite infestations, particularly in winter. Tailored advice on heating and ventilation habits can lead to valuable changes in hygrothermal conditions. In some cases, however, these improvements may be hindered by occupants' reluctance to change and/or by pre-existing adverse building conditions.

The following recommendations are made for similar future larger scale studies:

- Ventilation rates and air infiltration should both be measured.
- An adequate method for taking account of changes in weather conditions should be adopted.
- Occupant behaviour should be monitored, for example by utilising relevant diaries and/or measuring the frequency of window and fan usage.
- Selecting the same building types might facilitate assessing changes in occupant behaviours.
- Allergen and mite samples are invaluable, and the results should be expressed in terms of both allergen concentrations and total allergen content/load. Personal air samplers might also be useful.
- Hygrothermal and HDM population models are very useful to assess the likely effect of hygrothermal changes on mite infestations. Ideally, these models should be able to predict the effect of transient conditions.
- The placebo effect, the role of other allergens and the effectiveness of controls should be adequately addressed at the study design phase.

Acknowledgements

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References

1 National Academy of Sciences. Clearing the Air. Washington: National Academy Press, 2000.

2 Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I. A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae). Experimental and Applied Acarology 2006; 39: 127-148.

3 Cunningham MJ. Controlling Dust Mites Psychrometrically - a Review for Building Scientists and Engineers. Indoor Air 1996; 6: 249-258.

4 Arlian LG, Neal JS, Vyszenski-Moher DL. Fluctuating Hydrating and Dehydrating Relative Humidities Effects on the Life Cycle of *Dermatophagoides farinae* (Acari: Pyroglyphidae). Journal of Medical Entomology 1999; 36(4): 457-461.

5 Pike AJ, Cunningham MJ, Lester PJ. Development of Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) at constant and simultaneously fluctuating temperature and humidity conditions. Journal Of Medical Entomology 2005; 42(3): 266-269.

6 Luczynska C, Sterne J, Bond J, Azima H, Burney P. Indoor factors associated with concentrations of house dust mite allergen, Der p 1, in a random sample of houses in Norwich, UK. Clinical and Experimental Allergy 1998; 28(10): 1201-9.

7 Coates D (Twenty Twenty Television), Producer of: How to beat your kid's asthma. Screened on Channel 4 on the 6 and 10 April 2006.

10 Platts-Mills TAE, de Weck AL. Dust mite allergens and asthma - a worldwide problem. Journal of Allergy and Clinical Immunology 1989; 83: 416–27.

8 Sherman M. The use of Blower Door Data. Lawrence Berkeley Lab Report 35173. Berkeley: Lawrence Berkeley Laboratory, 1998.

9 Oreszczyn T, Pretlove SEC. Condensation Targeter II: Modelling surface relative humidity to predict mould growth in dwellings. Building Services Engineering Research & Technology 1999; 20(3): 143-153.

10 Arlian LG, Veselica MM. Effect of temperature on the equilibrium body water mass in the mite *Dermatophagoides farinae.* Physiology Zoology 1981; 54(4): 393-399.

11 Sanders C ed. IEA Annex 24. Heat, Air and Moisture transfer through new and retrofitted insulated envelope parts. Final Report. Vol. 2, Task 2 Environmental conditions. Belgium: Laboratorium Bouwfysica, 1996.

12 Howieson SG. Housing and asthma. Oxford: Taylor and Francis Ltd. Spon Press, 2005.

Table 1 Baseline measurements for building characteristics, hygrothermal conditions and mite infestation levels.

Dwelling	[θ]Moisture Product. (kg/day)	Volume (m^3)	[Δ]Air Infiltr. (ach^{-1})	*Mites (mites/ m^2)	*Der p1 Conc. (μg/g)	*Der p1 Load ($\mu g/m^2$)	#Pre, VPX (kPa)	#Pre, % Time CEH>RH (%)	#Pre, Temp (°C)	#Pre, RH (%)
1	7.2	163.1	0.2	20.3	23.0	1.16	0.6	92.1	20.9	68.7
2	4.2	198.6	0.4	0.0	1.7	0.10	0.2	18.1	20.7	52.3
3	3.4	127.2	0.5	0.0	0.3	0.13	0.4	66.8	20.8	57.3
5	6.5	484.5	0.9	0.0	0.4	0.01	0.2	0.0	20.9	40.4
6[c]	11.9	286.2	1.1	21.7	0.3	0.11	0.3	36.3	20.6	54.1
7	13.7	189.8	0.6	17.7	21.4	2.30	0.2	73.4	18.7	57.5
8[c]	6.4	137.4	0.5	0.0	3.3	0.24	0.4	37.4	22.3	55.8
9	7.9	215.9	1.4	0.0	(-)	0.02	0.2	47.2	20.2	54.5
10	6.3	141.3	0.6	0.0	0.9	0.11	0.4	20.2	22.1	54.6
11	10.1	396.1	1.3	5.3	1.8	0.31	0.2	99.2	17.4	61.8
12a	5.3	263.0	0.6	1.3	2.2	0.26	0.3	90.3	(17.6)	57.4
12b	5.3	263.0	0.6	0.0	0.8	0.12	0.3	100.0	(17.4)	60.5
Average	**7.7**	**227.3**	**0.7**	**5.1**	**1.4[α]**	**0.16[α]**	**0.3**	**56.8**	**20.5**	**55.8**
Outdoor Conditions									**11.4**	**76.8**

[c]Control Dwelling; *Bedroom, Average of: Mattress, Floor, Pillow; #Child Bedroom; [θ]Whole dwelling, estimated; [α]Geometric Mean; [Δ](Air-infiltration measured at 50 Pa)/20; (-) Missing data. Note: central heating in Dwelling 12 was malfunctioning in the pre-intervention study.

Table 2 Post-intervention hygrothermal results, and difference with pre-intervention conditions, for the child's bedroom.

Bedroom Number	Post: VPX (kPa)	Pre-Post# VPX (kPa)	Post: Temp. (°C)	Pre-Post# Temp. (°C)	Post: RH (%)	Pre-Post # RH (%)	Post: % Time CEH>RH	Pre-Post# % Time CEH>RH (%)
1	0.6	0.0	19.4	1.5	59.8	8.9	76.3	15.8
2	0.1	0.1	19.5	1.2	40.7	11.6	0.0	18.1
3	0.2	0.2	19.6	1.2	41.9	15.4	0.0	66.8
5	0.0	0.2	19.2	1.7	37.6	2.8	0.0	0.0
6	0.1	0.2	18.0	2.6	41.6	12.5	1.8	34.5
7	0.2	0.0	17.4	1.3	47.4	10.1	9.5	63.8
8	0.5	-0.1	21.9	0.4	48.4	7.4	0.1	37.3
9	0.2	0	21.3	-1.1	38.4	16.1	0.0	47.2
10	0.3	0.1	20.4	1.7	43.5	11.1	1.2	19.0
11	0.2	0	17.8	-0.4	47.8	14.0	12.1	87.1
12a	0.0	0.3	17.3	0.3	40.3	17.1	5.6	84.7
12b	0.2	0.1	17.2	0.2	47.1	13.4	2.8	97.2
Average*	**0.2**	**0.1**	**18.9**	**0.8**	**44.5**	**12.1**	**10.8**	**50.0**
Outdoor	**(n.a.)**	**(n.a.)**	**6.6**	**4.8**	**80.2**	**-3.4**	**(n.a.)**	**(n.a.)**

#Pre-Post Difference; *Excluding controls (bedroom 6 and 8)

Table 3 Pre-intervention study: predicted daily mite-growth risk (daily MPI)

Bedroom	Daily Bedroom MPI
1	1.03
2	0.97
3	0.98
4	0.94
4	0.97
5	1.00
6	0.95
7	0.98
8	0.95
9	0.97
10	0.98
11	1.00
12a	1.00
12b	0.99
Average	**0.98**

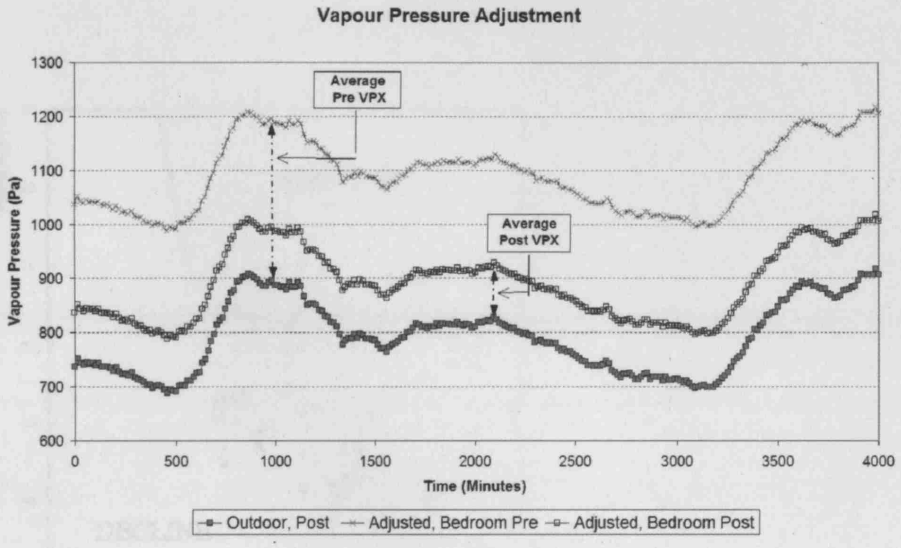

Figure 1: Schematic representation of the adjustment calculation for the vapour pressures, pre and post intervention.

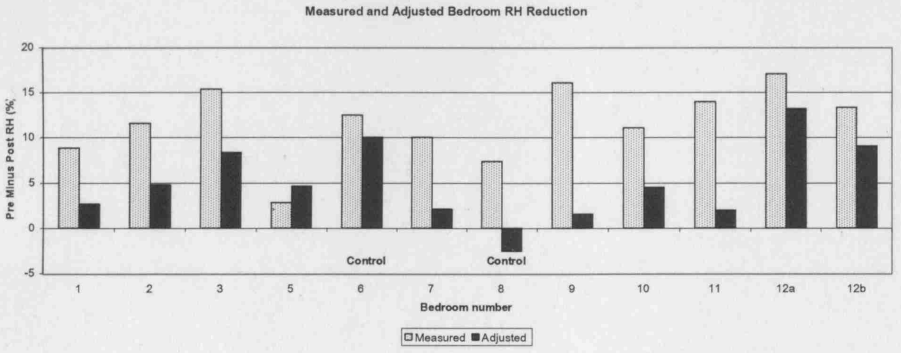

Figure 2: Measured and adjusted reduction in bedroom RHs.

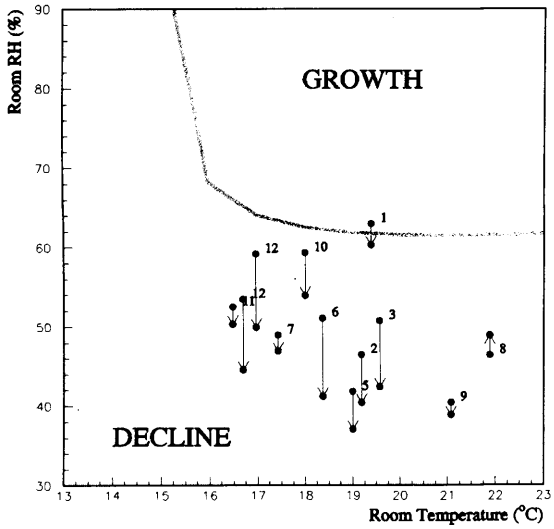

Figure 3: Predicted bedroom mite growth risk, using adjusted hygrothermal conditions: pre versus post intervention. The solid curve represents conditions where HDM populations are stable (MPI=1).

Volume 2: Appendix

The Psychrometric Control of House Dust Mites: Testing the Validity in UK Dwellings of Two Combined Hygrothermal Population Models for Beds

Marcella Ucci

The Bartlett School of Graduate Studies

University College London

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University College London

University of London

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Table of Contents for Volume 2 (Appendix)

Appendix to Chapter 2

A.2: House dust mites, atopy and asthma

Atopy is strongly associated with diseases such as asthma, hay fever, eczema and rhinitis, but not all atopic individuals develops clinical manifestations of allergy, nor everyone with a clinical syndrome compatible with allergic disease can be proven atopic when tested for specific IgE for common environmental allergens. This is particularly true for asthma (Jarvis and Burney, 1998). Asthma is the most serious of allergic diseases, being disabling and occasionally fatal. Many studies – especially cross-sectional - have demonstrated that asthma is strongly associated with atopy, particularly in children (Cole Johnson *et al.*, 2002). Therefore, a theoretical paradigm has often been advocated in which allergen exposure produces atopic sensitisation in susceptible individuals, and continued exposure then leads to clinical asthma through the development of airways inflammation, bronchial hyperresponsiveness and reversible airflow obstruction (Pearce *et al.*, 1999). However, not all asthma cases can be *attributed* to atopy, since the association between atopy and asthma may not reflect causality, at least in some cases. For example, inherited genetic factors could increase susceptibility both to asthma and to the production of raised IgE levels; higher total IgE levels could in part be a consequence of asthma itself (Pearce *et al.*, 1999). In this section the relationship between asthma and atopy is discussed further, with a particular focus on house dust mites.

Some authors have questioned the extent to which the development of asthma is attributable to atopy. Pearce *et al.* (1999) calculated the population attributable fraction for atopy and asthma, based on the review of several studies. The population Attributable Fraction (AF) can be defined as the proportion of disease cases over a specified time that would be prevented following the elimination of the exposures, assuming the exposures are causal (Rockhill *et al.*, 1998). If exposure (atopy in this case) has an odds ratio for asthma of R, the proportion of exposed cases (i.e. atopic asthmatics) that are attributable to exposure (i.e. atopy) is:

$AF_{exp}=(R-1)/R$

The proportion of all cases (i.e. asthmatics) in the general population that are attributable to exposure (atopy) is the population attributable fraction, which is:

$AF_{pop}=P*(R-1)/R$

where P is the proportion of all cases that are exposed (i.e. proportion of atopic asthmatics).

In their review Pearce *et al.* (1999) found that the proportion of cases attributable to atopy partly depends on the definition of atopy itself. If atopy is defined in a more stringent way (for example, four or more positive skin prick tests, rather than just one), the association with asthma *increases* (as reflected in the relative risk estimate), but since the proportion of atopic asthmatics decreases, the population attributable risk *decreases*. Pearce *et al.* concluded that the population-based proportion of asthma cases that attributable to atopy is usually less than 50%, varying from one third to two thirds, depending on the definition of atopy. However, Pearce *et al.* also emphasised that the studies may have differed methodologically, and they were not all carried out in the same population.

Sunyer *et al.* (2004) reported geographic variations in the effect of atopy on asthma in the European Community Respiratory Health Study (ECRHS). The ECRHS was the first international multicenter study in adults (20-44 years old) using a common standard protocol measuring atopy and asthma in the same time period (1990-1994). In the study, atopy was defined as the presence of IgE sensitisation to any allergen. The results from the study are summarised in Table A.2.1.

TABLE A.2.1 (Sunyer *et al.*, 2004) ECRHS (adults, aged 20-44): Prevalence of asthma and specific IgE by center (range in countries with 2 or more centers) and association (odds ratio or range of odds ratios in countries) between asthma and specific IgE at the individual level by center (number of individuals = 13,558; number of centers = 36)

Countries ordered by % of atopy* (no. of centers)	Prevalence, % (95% CI)					Odds ratio (95% CI)↔			
	Asthma	House dust mite	Cat	Timothy Grass	Atopy*	House dust mite	Cat	Timothy Grass	Atopy*
Estonia (1)	7	9	5	9	18	1.82	8.74	3.12	1.25
Iceland (1)	3	9	7	12	23	8.91	7.02	4.59	4.21
Spain (5)	4-11	7-28	3-13	9-20	17-42	1.48-4.54	2.78-8.90	1.62-4.02	1.33-5.44
Norway (1)	7	14	7	15	26	3.17	5.46	2.76	5.16
Italy (3)	6-15	11-13	4-7	12-21	24-30	2.53-5.30	1.10-9.51	2.76-5.42	2.94-4.85
Sweden (3)	8-10	7-12	13-14	17-18	30-32	1.88-2.36	2.60-5.54	2.02-3.58	1.92-5.17
France (4)	6-13	18-35	7-10	12-20	29-43	1.79-4.64	3.43-6.48	1.37-3.98	1.53-4.60
Belgium (2)	5-9	22-27	9-9	16-17	35-36	3.65-3.65	2.78-5.03	4.17-5.10	4.24-5.28
Germany (2)	3-7	16-19	8-11	21-25	35-40	0.23-2.55	2.60-4.47	1.35-2.55	1.36-3.31
United Kingdom (4)	9-14	20-28	8-14	13-27	34-44	2.01-5.07	2.33-5.17	1.62-2.86	2.04-3.93
The Netherlands (3)	5-7	24-29	6-10	17-22	36-41	2.06-6.14	3.75-5.52	2.44-5.49	2.03-5.74
Ireland (1)	12	35	7	17	41	3.15	3.62	5.51	2.07
New Zealand (3)	11-14	31-33	6-13	23-33	40-46	1.74-6.14	0.83-8.34	2.19-3.14	1.57-4.58
United States (1)	12	19	13	34	43	1.01	2.13	2.48	2.52
Switzerland (1)	10	19	15	33	45	1.86	1.31	1.75	1.53
Australia (1)	12	32	9	29	45	2.89	3.24	2.41	3.22
All (95% CI),	9 (8-10),	21 (18-23),	8 (7-10)	19 (17-21),	34 (31-37),	2.78	4.18	2.63	2.82
#p value for heterogeneity	<.001	<.001	<.001	<.001	<.001	(2.41-3.20), .14	(3.54-4.93), .45	(2.30-3.02), .92	(2.44-3.28), .15

*Any: house dust mite, cat, timothy grass, C herbarum, and birch, P judaica, or ragweed. #Estimated with meta-analysis. ↔Adjusting for age, sex, and smoking.

The results show that the prevalence of specific IgE sensitisation varied widely between centres, even within the same country (e.g. dust mites in Spain or in France). The association between IgE sensitisation and asthma was strong, but the associations were not statistically significantly heterogeneous across centres, for any allergen. Sunyer *et al.* also calculated the proportion of asthma cases attributable to atopy and to specific allergens in the various centres. The results are summarised in Table A.2.2.

Table A.2.2 (Sunyer *et al.*, 2004). ECRHS (adults, aged 20-44): AF of asthma, defined on the basis of symptoms, caused by specific IgE sensitization and atopy by center

Countries ordered by % of atopy*	Center	HDM	Cat	Tim. grass	Atopy* (95% CI)
Estonia	Tartu	6	17	13	4 (219.0 to 22.0)
Iceland	Reykjavik	35	28	25	40 (22.1 to 64.5)
Spain	Albacete	3	9	7	11 (24.8 to 24.8)
	Oviedo	10	6	15	25 (25.9 to 46.6)
	Galdakao	40	23	13	45 (0.0 to 70.2)
	Huelva	14	22	10	9 (227.3 to 35.5)
	Barcelona (bcn)	32	37	8	61 (227.8 to 88.1)
Norway	Bergen	19	19	18	47 (26.1 to 61.3)
Italy	Pavia	24	0	24	26 (26.1 to 47.8)
	Turin	10	17	20	37 (10.7 to 55.2)
	Verona	21	21	21	44 (6.7 to 66.8)
Sweden	Umea	6	31	26	50 (25.3 to 66.5)
	Goteborg	8	15	13	28 (5.2 to 44.7)
	Uppsala	7	16	20	20 (25.9 to 39.2)
France	Grenoble	12	15	13	16 (216.0 to 39.7)
	Paris	16	18	21	36 (14.4 to 51.6)
	Montpellier	15	11	5	12 (28.8 to 28.3)
	Bordeaux	48	25	23	55 (33.1 to 69.4)
Belgium	South-Antwerp	31	11	27	46 (0.7 to 70.9)
	Antwerp city	37	22	31	55 (17.9 to 75.4)
Germany	Erfurt	214	10	7	11 (226.4 to 37.4)
	Hamburg	19	22	24	43 (19.5, 60.0)
United Kingdom	Cardiff	19	11	11	22 (21.6 to 40.2)
	Ipswich	36	22	23	44 (18.1 to 61.3)
	Norwich	19	20	17	26 (22.2 to 45.9)
	Cambridge	29	12	12	38 (213.8 to 66.6)
The Netherlands	Groningen	54	20	23	58 (13.5 to 79.7)
	Bergen op Zoom	20	15	20	36 (2.1 to 57.7)
	Gellen	19	14	39	26 (217.3 to 53.8)
Ireland	Dublin	35	12	31	26 (29.7 to 50.2)
New Zealand	Hawkes-Bay	14	21	23	14 (229.7 to 43.0)
	Wellington	51	17	18	52 (23.7 to 70.2)
	Christchurch	51	29	30	49 (16.3 to 68.7)
United States	Portland	0	10	29	35 (3.8 to 56.2)
Switzerland	Basel	12	4	17	17 (210.2 to 37.2)
Australia	Melbourne	32	13	25	45 (21.2 to 61.2)
ALL#		18.2 (13.7, 22.4)	14.1 (11.8, 16.3)	17.1 (14.0, 20.1)	30.4 (24.9 to 35.5)
p value for heterogeneity		<.001	.30	.91	.012

*Atopy: IgE sensitization to any of house dust mite, cat, timothy grass, C herbarum, and birch, P judaica, or ragweed.
#AF in the 36 centers estimated with meta-analysis.

The results in Table A.2.2 show that the population attributable fraction of asthma caused by atopy is approximately 30%. The AF_{pop} for atopy could have been underestimated because of the definition of asthma used. The overall AF_{pop} for atopy increased to 42.6% when the diagnosis of asthma was based on wheezing and bronchial hyperresponsiveness, to 45.3% with physician-diagnosed asthma, and 47.9% when patients reported more than 12 attacks of asthma within the past year. The overall AF_{pop} for sensitisation to dust mites was 18.2%. The AF_{pop} for dust mites varied significantly between centres (p value for heterogeneity < 0.001). Sunyer *et al.* concluded that if total elimination from a given population of sensitisation to the allergen considered in their study could be obtained - which is unrealistic – this might possibly result in a reduction of 30% of prevalent asthma cases. Although 56% of asthmatics are sensitised to common aeroallergens, the AF indicates that around 60% of them (i.e. AF among the exposed) would have been prevented by preventing any sensitisation to occur. Sunyer *et al.* highlighted that only geographic variations in the AF for house dust mites were statistically heterogeneous, suggesting that the prevalence of sensitisation to dust mites is the main determinant of geographic variations in the population fraction of asthma attributable to atopy. Sunyer *et al.* conclude that "*IgE sensitisation to common allergens has an effect on asthma prevalence, which varies widely amongst centres. Reasons for the wide variation remains unknown, but they do not seem to be due to the strength of the association between atopy and asthma. Most important appear to be other exposures influencing the expression of asthma among atopic and non-atopic individuals. Levels of allergens in the environment as reflected by the prevalence of atopy – particularly dust mites – also seem to play a role. The present results reinforce the idea that atopy is only one factor in the constellation of factors that play a role in asthma prevalence*". Sunyer *et al.* also point out that interpretation of their results in terms of causality must be taken with caution, since in their study the AF refers to asthma *prevalence* (proportion of *existing* cases, as opposed to *incidence*, i.e. *new* cases). Therefore, their findings are unable to determine whether sensitisation to allergens plays a role in asthma *development*, but only in asthma *prevalence*.

Jaakkola *et al.* (2006) examined the relationship between sensitisation to mites and moulds, and asthma *development* in adults. The study was a population-based incident case-control study, carried out in Finland on adults (21-63 years old) with a total of

485 cases (clinical asthma diagnosis) and 665 controls. The authors found that specific IgE antibodies to *Dermatophagoides Pteronyssinus* and *Acarus Sirus* were not very common, but when detected they were related to significantly increased risk of incident asthma (Odds Ratio for *Dermatophagoides Pteronyssinus*: 2.3; 95% CI: 1.51-3.49). The risk of new asthma increased with increasing IgE antibody levels to *Dermatophagoides Pteronyssinus* (and *Aspergillus fumigatus*). Jaakkola *et al.* also found that the fraction of new adult-onset asthma attributable to IgE antibodies for common aeroallergens was 66% (95% CI, 55-74) among atopic cases. The attributable fraction in the whole working-age population was 30% (95% CI, 23-36). These findings are not dissimilar to those found by Sunyer *et al.* (2004) or by Pearce *et al.* (1999), although they studied the relationship between atopy and asthma *prevalence* in adults.

Carroll *et al.* (2006) studied a total of 400 children (7-18 years) with asthma in the UK (North Staffordshire and Sheffield). The authors concluded that increasing atopic sensitisation is associated with increasing disease *severity* in children with asthma.

Allergic sensitisation and family history of asthma as risk factors for asthma onset were also studied by Backlund *et al.* (2006). A cohort study followed 3525 Swedish children aged 7-8 years old in 1996, until they were 11-12 years old. The study found that sensitisation to any allergen (*Dermatophagoides Pteronyssinus*, *Dermatophagoides Farinae, Cladosporium* and *Alternaria*) was the strongest risk factor for current asthma (OR: 4.88, 95% CI 3.31-7.20) at age 7-8, increasing to OR 5.63 (95% CI 3.88-8.18) at age 11-12 with no difference between sexes. A family history of asthma was the second strongest risk factor with OR 3.04 (95% CI 2.07-4.47) at age 7-8 and OR 2.78 (95% CI 1.96-3.94) at age 11-12. The study also examined asthma remission, defined as subject reporting current asthma at one occasion, and reporting neither wheeze nor medication use in the past 12 months during the next occasion. Remission was found inversely correlated with allergic sensitisation. However, it should be mentioned that a study with a longer timescale should be carried out in order to confirm the figures on remission.

The studies described so far show that atopy plays a role in asthma persistence (prevalence), incidence and severity. There are significant geographic variations in asthma prevalence, but the strength of the association between asthma and atopy does not appear to vary significantly worldwide in adults. However, the population fraction

of asthma attributable to atopy varies in relation to the definition of asthma and of atopy. Furthermore, the population fraction of asthma attributable to dust mite sensitisation does appear to vary significantly worldwide – at least in adults - suggesting that the prevalence of sensitisation to dust mites (and exposure to dust mite allergen) is the main determinant of geographic variations in the population fraction of asthma attributable to atopy. In England and Wales the (adult) population fraction of (prevalent) asthma attributable to dust mite sensitisation varies from 19% to 36%. This means that 19-36% of adult asthma could be potentially prevented, if HDM sensitisation could be avoided. This raises the following questions: a) is there a dose-response relationship between HDM allergen exposure and sensitisation/asthma? b) are there threshold levels of HDM allergen exposure, below which sensitisation does not occur?.

The Committee on the Assessment of Asthma and Indoor Air, from the Institute of Medicine of the National Academy of Sciences (US) concluded that: "*There is sufficient evidence of a causal relationship between HDM allergen exposure and exacerbation of asthmatics specifically sensitized to dust mites. Continual exposure to dust mite allergens is also a contributing cause of chronic bronchial hyperreactivity. There is sufficient evidence of a causal relationship between dust mite allergen exposure and the development of asthma in susceptible children*" (National Academy of Sciences, 2000). The Committee specifies that 'causality' is not intended it in its old-fashioned concept of sufficient *and* necessary cause (*sufficient* cause: all persons exposed to x will develop asthma; *necessary* cause: all cases of asthma are caused by x). Rather, the Committee pointed out that it is generally recognized that most health outcomes of interest have multifactorial etiologies. Therefore, causality occurs if there is at least one person whose asthma was caused by a certain factor X.

Several studies support the notion that dust mites play a major role in asthma exacerbation and development in susceptible individuals. However, some authors are much more cautious (Pearce *et al.*, 2000). One of the issues under discussion is not necessarily whether exposure to dust mite allergen can "cause" asthma in at least one person, but rather whether HDM exposure causes asthma for a *significant* proportion in the asthmatics population. In the previous section it was highlighted that the population fraction of asthma attributable to dust mite sensitisation varies significantly worldwide – at least in adults - suggesting that the prevalence of

sensitisation to dust mites (and exposure to dust mite allergen) is the main determinant of geographic variations in the population fraction of asthma attributable to atopy (Sunyer *et al.*, 2004).

When considering the relationship between HDM allergens, atopy and asthma, a number of interrelated issues have to be considered:

1. Does exposure to HDM allergen increase the risk of *specific sensitisation* (in at risk individuals), and if so, is there a (linear) dose-response relationship between allergen exposure and sensitisation?
2. Does exposure to HDM allergen increase the risk of *asthma onset* (in at risk individuals?), and if so, is there a (linear) dose-response relationship between allergen exposure, HDM sensitisation and asthma onset?
3. Does exposure to HDM allergen increases the *severity of asthma symptoms* in sensitised individuals?
4. Can threshold levels for HDM allergen exposure be determined, above which the following occur: a) HDM sensitisation; b) asthma development; c) asthma exacerbation?

These issues are very important when trying to formulate strategies for: reducing symptoms in asthmatic patients, reducing the incidence of HDM sensitisation and reducing the incidence of asthma.

In 1989 a team of experts discussed the worldwide problem of dust mite allergens and asthma in an International Workshop under the auspices of the WHO (Platts-Mills and de Weck, 1989). One of the main conclusions of the workshop was the provisional recommendation of threshold levels for mite-allergen exposure. The experts proposed that a level of 2 μg of Der p1 per gram of dust (equivalent to 100 mites per gram) should be regarded as a risk factor for sensitisation and the development of asthma. The higher level of 10 μg of Der p1 per gram of dust (equivalent to 500 mites per gram) was proposed as a major risk factor for the development of acute asthma in mite-allergic individuals. In 1992, a second International Workshop took place, which confirmed the threshold levels and the dose-response relationship between HDM allergen exposure, HDM sensitisation and asthma development (Platts-Mills *et al.*, 1992). These recommended levels are often referred to in the literature as the "WHO threshold levels" for dust mite allergens and asthma.

However, since the formulation of the "WHO" threshold levels, further research has been carried out, which provides additional insight into the role of HDM allergen exposure on asthma, as well as into the exposure thresholds. Indeed, a *third* International Workshop of experts on dust mites and asthma took place in 1997, highlighting that the pattern of sensitisation to specific allergens reflects the *mean level* of allergen found in the houses of those communities where the patients live. The experts concluded that there was still sufficient evidence for a dose-response relationship between exposure to mite allergens and sensitisation to these allergens. They also highlighted that mite sensitisation is a major independent risk factor for asthma in New Zealand, coastal Australia, Florida, central Virginia. However, in other countries such as Scandinavia and the mountain states of the US, sensitisation to domestic animal allergens may have the strongest association with asthma. The experts therefore slightly changed their recommendations on threshold levels, concluding that in areas where the *mean level* of dust mite group I allergen in houses is 2 μg of Der p1 per gram of dust or more, sensitisation to mites has consistently been found to be associated with asthma. In the report of the third International Workshop there was no mention of the threshold of 10 μg of Der p1 per gram of dust. This is because the experts concluded that the relationship between HDM allergen exposure and asthma symptoms is complex, which makes the identification of a threshold level in HDM allergen exposure for asthma exacerbation quite difficult (Platts-Mills *et al.*, 1997).

Although a single threshold of HDM allergen exposure for exacerbation of asthma symptoms may be difficult to assess, some studies have correlated disease severity and allergen exposure. For example, Custovic *et al.* (1996) concluded that clinical activity and severity of asthma in mite-sensitive non-smoking adult patients is related to mite allergen exposure, with levels in beds being an important indicator that correlated with disease activity.

In identifying the threshold levels for mite sensitisation, the reports of the International Workshops did not explicitly make a distinction between 'at risk individuals' (i.e. with a family history of atopy and/or asthma) and 'not at risk individuals'. However, Custovic and Chapman highlighted that most studies which had investigated the relationship between exposure and sensitisation up to that time had focused on individuals at risk of developing atopy (Custovic and Chapman,

1998). Therefore, Custovic and Chapman concluded that exposure to 2 μg of Der p1 per gram of dust or more should be regarded as a risk factor for the development of mite-specific IgE antibody and asthma in *susceptible* children. This value is applicable to the population and should not be extrapolated to the clinical situation and to individual patients. Custovic and Chapman also concluded that a simple threshold level for provocation of asthmatic symptoms had not been yet identified. In a discussion paper, Marks also concluded that it seems likely that a threshold, below which sensitisation does not occur, is either much lower than 2 μg of Der p1 per gram of dust, or does not exists (Marks, 1998). However, Marks also concluded that there does appear to be an upper limit above which further increases in exposure do not cause any further increase in risk of sensitisation. Marks suggested that this upper limit is approximately 10 μg of Der p1 per gram of dust. In populations such as children in Sydney where virtually everyone is exposed to HDM levels > 10 μg of Der p1 per gram of dust, other factors influence the risk of developing allergy. Marks therefore concludes that HDM exposure is a necessary but not a sufficient factor in the development of HDM sensitisation.

The main conclusions emerging from reports such as those produced by the International Workshops on dust mites and asthma (Platts-Mills *et al.*, 1989, 1992, 1997) were based on the hypothesis that exposure to high levels of HDM allergen during early childhood contributes to HDM sensitisation and that continued exposure causes airway inflammation leading to the development of asthma in children. However, most studies carried out in the 1990s were either cross-sectional or case-control, where it is difficult to assess the role of HDM allergen exposure on asthma onset. A fundamental study on the role of HDM exposure and asthma onset in children was a *cohort* study of 67 British children at risk for allergic disease because of family history, where the relationship between Der p1 exposure and the *development* of sensitisation and asthma was investigated (Sporik *et al.*, 1990). The study concluded that there was a trend towards an increasing degree of sensitisation at the age of 11 with greater exposure at the age of 1 ($p=0.062$). Also, the relative risk of asthma development was 4.8 ($p=0.05$) for exposures to more than 10 μg of Der p1 per gram of dust.

The study by Sporik *et al.* has often been quoted in subsequent studies (included those produced by the WHO International Workshops) as proof of a causal relationship

between HDM exposure and childhood asthma onset. However, the study had some limitations: a fairly small sample size, the study focussed on at risk children (family history), and it was carried out in an area with relatively high levels of mite allergen. Subsequent cohort studies have presented a more contradictory picture on the role of HDM exposure in the onset of childhood asthma. In the German Multicentre Allergy Study, 939 newborn infants from 5 German cities were followed for 7 years (Lau *et al.*, 2000). The study was unable to find a consistent dose-response relationship for early indoor allergen exposure and "doctor's diagnosed asthma", "wheezing within the last 12 months", or "wheezing ever". However, sensitisation to mite and cat allergens was associated with indoor allergen exposure and with wheezing. The author of the study did highlight that the allergen levels found in carpets in their study were fairly low. Lau *et al.* conclude that whereas allergen exposure has a clear influence on atopy, in areas with moderate exposures the link to asthma is less pronounced.

A subsequent cohort study based in the UK established different conclusions from Sporik *et al.* (1990), and from Lau *et al.* (2000). Cullinan et al. (2004) followed 625 children in Ashford (Kent, UK) from birth to the age of 5.5 years, at which time 552 underwent skin prick testing for HDM and cat. When the impact of allergen exposure on sensitisation and on atopic wheeze was assessed (Fig. A.2.1), it was found that the exposure-response relationship for each allergen was neither linear nor monotonic, but showed an increase in risk at low levels of exposures, followed by a flattening or, in some cases, a reduction of risk at higher exposures. Different patterns were observed in relation to family history (atopic father) and birth order (first born).

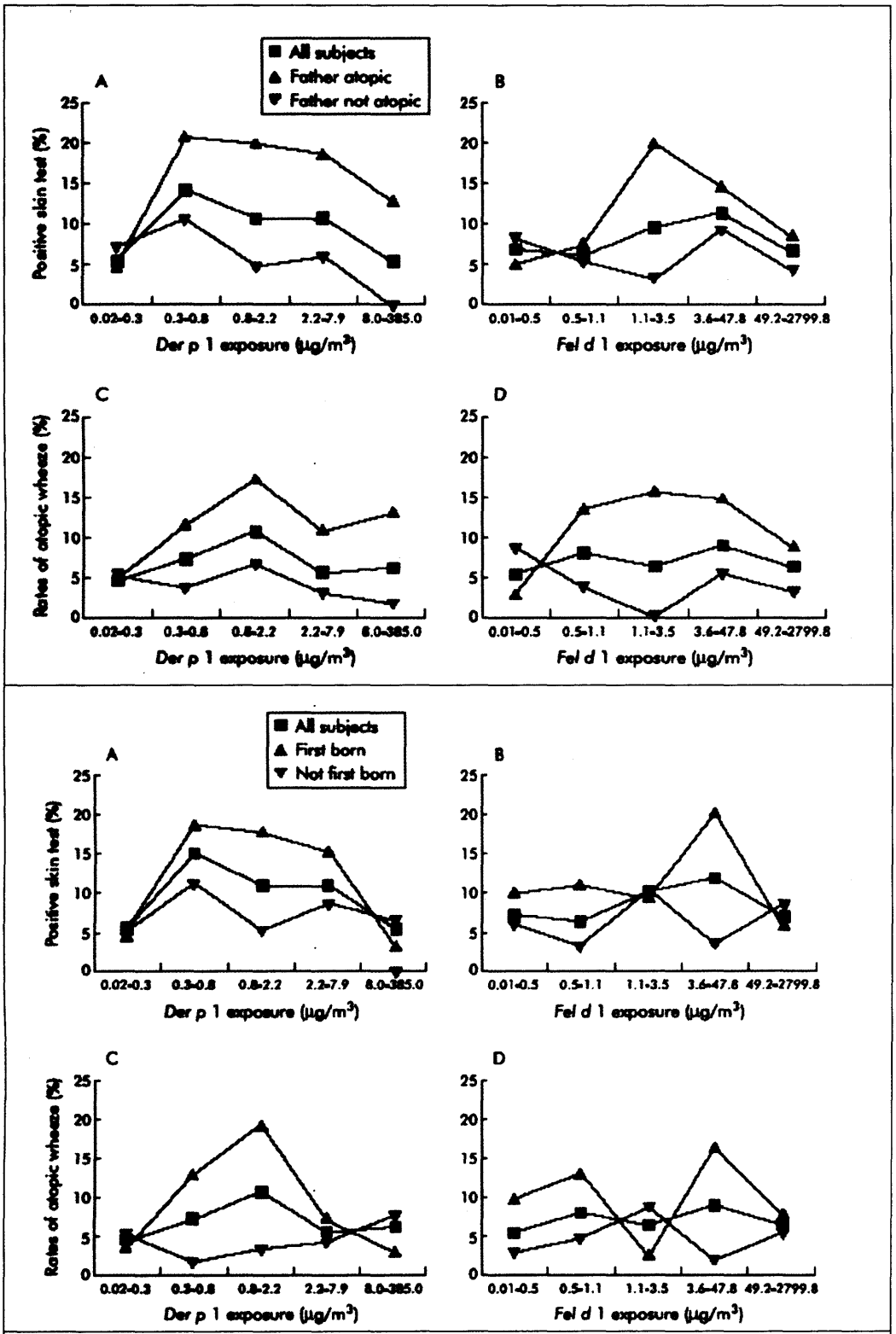


Fig A.2.1 Relationships between exposure to Der p1 or Fel d1, and sensitisation or atopic wheeze, in a cohort of British children (Cullinan *et al.*, 2004).

Another cohort study (Childhood Allergy Study) carried out in the US (Detroit) on 428 children from birth up to the age of 6 or 7 also confirmed that family history of atopy can be an effect-modifier in the exposure-response relationships between HDM allergen, atopy and asthma onset. They found that where absence of parental history of allergic disease appeared to decrease the risk HDM sensitisation at the age of 6 or 7, whereas the risk was increased in children with a parental history of allergic disease (Cole Johnson *et al.,* 2004).

The results obtained by Cole Johnson *et al.* were not reproduced by another cohort study carried out in the Netherlands (Brussee *et al.*, 2005). The Prevention and Incidence of Asthma and Mite Allergy (PIAMA) study investigated the effect of allergen exposure at 3 months of age on the development of sensitisation, wheeze, and physician diagnosed asthma in the first 4 years of life in a birth cohort of 3291 children. The results were stratified by maternal atopy. The results from PIAMA study on allergen exposure and specific sensitisation agree with the findings of a dose-response relationship found by the German study from Lau *et al.* (2000). However, both studies had low mite allergen levels. The PIAMA study results did not agree with those from the US Childhood Allergy Study (Cole Johnson *et al.*, 2004), where parental history was an effect-modifier. However, it should be emphasised that in the PIAMA study, dust samples were taken from the child's bed (which was new in 1/3 of the cases), while in the other studies - Lau *et al.*, 2000; Cullinan *et al.*, 2004; Cole Johnson *et al.*, 2004 - the samples were taken from carpets (living room for Cullinan *et al.*). The authors of the PIAMA study also highlighted that the cohort needed to be followed further, in order to assess the long-term consequences of early life exposures.

The issue of dose-response relationship between exposure to dust mite allergen and HDM sensitisation was also addressed by the PARSIFAL study, a cross-sectional study carried out in 5 European countries: Sweden, Switzerland, Germany, Austria and the Netherlands (Schram-Bijkerk *et al.*, 2006). The study included children aged 5-13 years, from a randomly selected population of 229 children from livestock farms, 122 Steiner children, with respectively 60 and 67 control children, with nearly equal numbers per country. The results showed that mite allergen levels were in the same order of magnitude for all groups of children. However, farm children had a lower prevalence of mite sensitisation. In the PARSIFAL study highest sensitisation rates

were observed in the intermediate exposure group, consistently across farm, Steiner and reference children. In order to study the dose-response relationship between HDM allergen exposure and HDM sensitisation, as well as the effect of microbial agents on such relationship, smoothed dose-response curves were determined by generalised cross validation. The curves were stratified by each microbial agent and dichotomised according to the median level of each agent (Figure A.2.2).

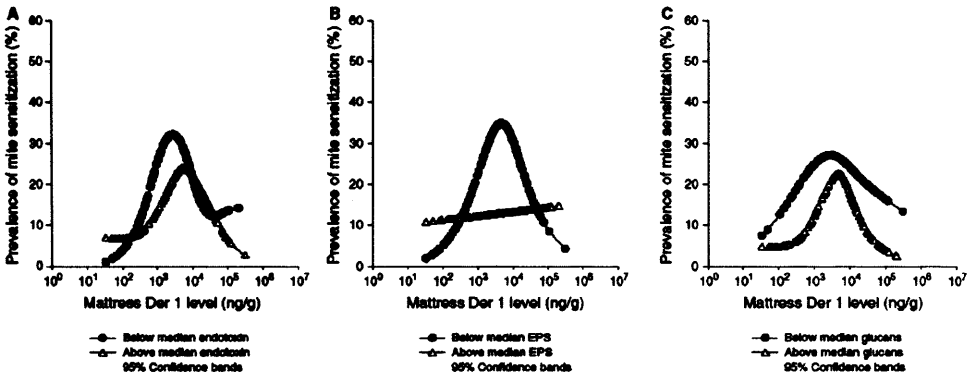


Fig A.2.2 Smoothed plots of the prevalence of mite sensitisation in relation to mite allergen levels, stratified by: A) dichotomised endotoxin levels; B) dichotomised extracellular polysaccharides (EPS) levels; C) dichotomised glucan levels in mattress dust (Schram-Bijkerk *et al.*, 2006).

Figure A.2.2 shows that for both endotoxin and glucan, the curve is bell-shaped below and above median levels. However, for EPS the curve is bell-shaped below median EPS levels but the curve is very much flattened above median EPS levels. The authors highlight that their results are not in line with previous cohort studies showing a linear dose-response relationship between mite allergen levels and mite sensitisation (e.g. Lau et al., 2000). The author of the PARSIFAL study suggest that this might be due to differences in allergen levels - which were rather high in their study – and/or in the study population. The results from the PARSIFAL study are in part similar to those found by Cullinan *et al.* (2004) in a UK cohort, where an increased risk of sensitisation was found at low allergen levels, and an attenuated risk was found at high levels. However, in Cullinan *et al.*, as well as in Cole Johnson *et al.* (2004), parental history of allergy appeared to be an effect-modifier for HDM allergen exposure and HDM sensitisation - although the relationship were not statistically significant - while this effect-modification was not observed in the PARSIFAL study.

The Asthma Multicenter Infant Cohort Study (AMICS) followed prospectively a representative population for 3 European centres: Ashford (Kent, UK), Menorca Island and Barcelona (Spain) (Torrent *et al.*, 2006). They aimed to assess the role of early exposure to Der p1 and Fel d1 in sensitisation at the age of 4 years. The results showed that the exposure profiles among centres were very different, with high Der p1 levels in Menorca and high Fel d1 levels in Ashford. However, the proportion of children sensitised to Der p1 did not differ greatly among centres. Figure A.2.3 and A.2.4 show the variations in sensitisation rates at the age of 4 years by Der p1 and Fel d1 levels in early infancy, after adjustment for confounding variables. The figure shows that Der p1 levels had a positive association with sensitisation for Barcelona only.

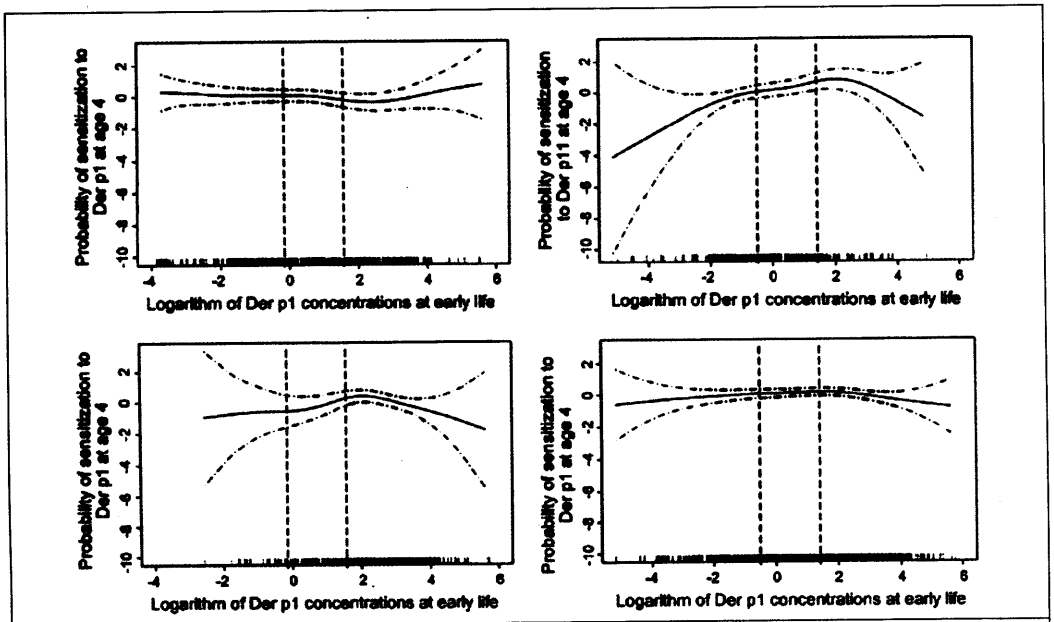


Fig A.2.3 Probability (logit scale) of sensitisation to house dust mite allergen (Der p1) at age 4 years and dust mite allergen exposure in early life. Upper left graph: *Ashford; upper right graph: Barcelona; lower left graph: Menorca; lower right graph: all centres.* Vertical line indicates tertile cut-off points. (Torrent *et al.*, 2006)

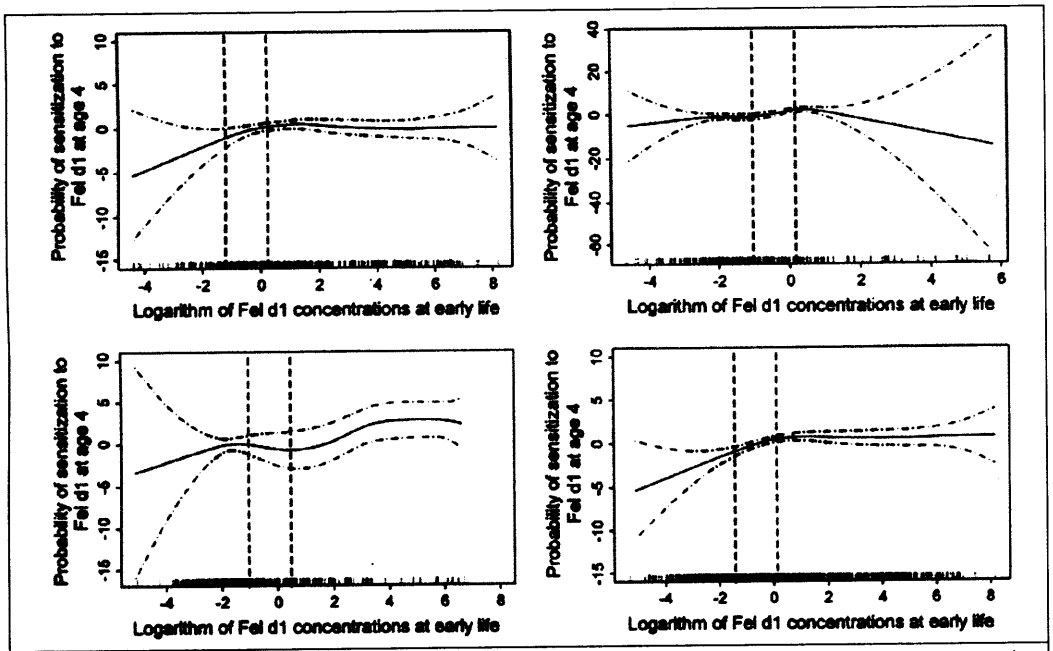


Fig A.2.4 Probability (logit scale) of sensitisation to cat allergen (Fel p1) at age 4 years and cat allergen exposure in early life. Upper left graph: *Ashford; upper right graph: Barcelona; lower left graph: Menorca; lower right graph: all centres.* Vertical line indicates tertile cut-off points. (Torrent *et al.*, 2006)

Pooled multiple regression models for Der p1 indicated that only maternal atopy and male sex had a statistically significant positive association with Der p1 sensitisation. There was no relationship with dust mite allergen exposure in early life and HDM sensitisation. A threshold level for sensitisation could not be found: even at a concentration of less than 0.032 μg Der p1/g of dust, 2 out of 20 children were sensitised. In summary, the authors of the AMICS study conclude that the dose-response relationships between allergen exposure and sensitisation differ between allergen and between geographical areas. The relationship between allergen exposure and sensitisation might not be linear.

This appendix discussed the role of house dust mites on HDM sensitisation, asthma development and asthma severity, with a focus on threshold levels of exposure. In summary, there is little doubt that exposure to HDM allergen leads to exacerbation of asthma symptoms in susceptible individuals, and that asthma severity is greater with greater exposure to HDM allergens. However, no single threshold level of HDM allergen exposure can be identified for exacerbation of asthma symptoms. Exposure to HDM allergen may lead to HDM sensitisation and to asthma development. However, there is some controversy on the extent of asthma onset which can be attributed to HDM exposure. Furthermore, there is some contradictory evidence on the relationship between exposure to HDM allergens, and HDM sensitisation or asthma onset. This is mostly because these relationships can vary in relation to: study population (i.e. genetic factors and typical exposure levels), family history of asthma/atopy, and other confounding factors – particularly the presence of potentially "protective" factors in the environment, such as endotoxins or extracellular polysaccharides (EPS). Because of these factors, it is unwise to identify a threshold level of HDM exposure for sensitisation or asthma onset, which can be applied to any population. Some evidence suggests that in England and Wales the (adult) population fraction of (prevalent) asthma attributable to dust mite sensitisation varies from 19% to 36%. Some evidence also suggests that in some study populations HDM sensitisation might occur at intermediate exposure levels, with a bell-shaped relationship (possibly modified by other potentially "protective" factors). This should be taken into account in any primary prevention studies aiming at reducing HDM allergen levels for the reduction of HDM sensitisation.

Appendix A.2: References

Backlund AB, Perzanowski MS, Platts-Mills T, Sandstrom T, Lundback B, Ronmark E, (2006), Asthma during the primary school ages - prevalence, remission and the impact of allergic sensitization, *Allergy*, 61(5): 549-55.

Brussee JE, Smit HA, Van Strien RT, Corver K, Kerkhof M, Wijga AH, Aalberse RC, Postma D, Gerritsen J, Grobbee DE, De Jongste JC, Brunekreef B, (2005), Allergen exposure in infancy and the development of sensitization, wheeze, and asthma at 4 years, *The Journal Of Allergy And Clinical Immunology*, 115 (5): 946-952.

Carroll WD, Lenney W, Child F, Strange FC, Jones PW, Whyte MK, Primhak RA, and Fryer AA, (2006), Asthma severity and atopy: how clear is the relationship?, *Archives of Disease in Childhood*, 91:405-409.

Cole Johnson C, Ownby DR, Zoratti EM, Alford SH, Williams KL and Joseph CLM, (2002), Environmental epidemiology of pediatric asthma and allergy, *Epidemiologic Reviews*, 24: 154-175.

Cullinan P, MacNeill SJ, Harris JM, Moffat S, White C, Mills P and Newman Taylor AJ, (2004), Early allergen exposure, skin prick responses, and atopic wheeze at age 5 in English children: a cohort study, *Thorax*, 59:855-861.

Custovic A and Chapman M, (1998), Risk levels for mite allergens. Are they meaningful?, *Allergy*, 53(Suppl. 48):71-76.

Custovic A, Taggart SC, Francis HC, Chapman MD, Woodcock A, (1996), Exposure to house dust mite allergens and the clinical activity of asthma, *Journal of Allergy and Clinical Immunology*, 98(1): 64-72.

Jaakkola MS, Ieromnimon A, Jaakkola JJK, (2006), Are atopy and specific IgE to mites and molds important for adult asthma?, *Journal of Allergy and Clinical Immunology*, 117 (3): 642-648.

Jarvis D, Burney P, (1998), The epidemiology of allergic disease, *BMJ*, 316: 607-610.

Lau S, Illi S, Sommerfeld C, Niggemann B, Bergmann R, von Mutius E, Wahn U, and the Multicentre Allergy Study Group, (2000), Early exposure to house-dust mite

and cat allergens and development of childhood asthma: a cohort study, *The Lancet*, 356(9239): 1392-1397.

Marks GB, (1998), House dust mite exposure as a risk factor for asthma: benefits of avoidance, *Allergy*, 53(Suppl. 48): 108-114.

National Academy of Sciences (Committee on the Assessment of Asthma and Indoor Air, Institute of Medicine, U.S.), (2000), *Clearing the Air,* Washington: National Academy Press.

Pearce N, Douwes J, Beasley R., (2000), Is allergen exposure the major primary cause of asthma?, *Thorax*, 55(5): 424-31.

Pearce N, Pekkanen J, and Beasley R, (1999), How much asthma is really attributable to atopy?, *Thorax*, 54: 268–272.

Platts-Mills T and de Weck A, (chairmen), (1989), Dust mite allergens and asthma – a worldwide problem. Report of an International Workshop, *Journal of Allergy and Clinical Immunology*, 83(2, part 1): 416-427.

Platts-Mills TA, Thomas WR, Aalberse RC, Vervloet D, Chapman MD, (1992), Dust mite allergens and asthma: report of a second international workshop, *Journal of Allergy and Clinical Immunology*, 89(5): 1046-60.

Platts-Mills TA, Vervloet D, Thomas WR, Aalberse RC, Chapman MD, (1997), Indoor allergens and asthma: report of the Third International Workshop, *Journal of Allergy and Clinical Immunology*, 100(6 Pt 1): S2-S24.

Rockhill B, Newman B, Weinberg C, (1998), Use and Misuse of Population Attributable Fraction, *American Journal of Public Health*, 88(1): 15-19.

Schram-Bijkerk D, Doekes G, Boeve M, Douwes J, Riedler J, Üblagger E, von Mutius E, Budde J, Pershagen G, van Hage M, Wickman M, Braun-Fahrländer C, Waser M, Brunekreef B, the PARSIFAL study group, (2006), Nonlinear relations between house dust mite allergen levels and mite sensitization in farm and nonfarm children, *Allergy*, 61(5): 640-647.

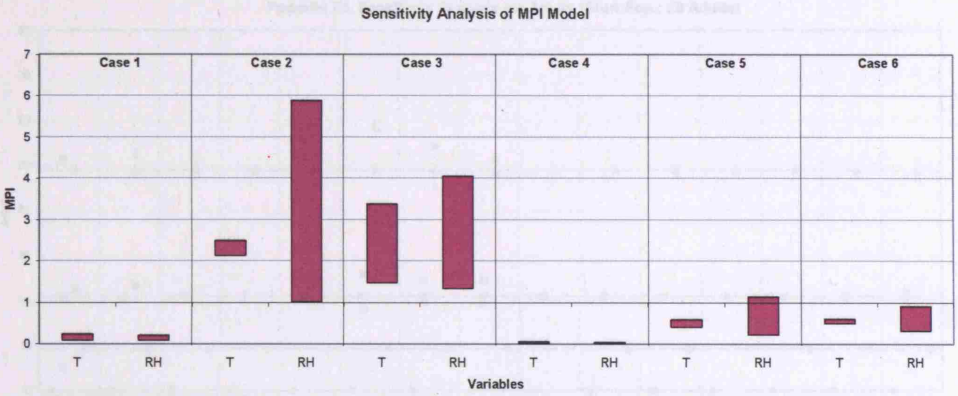
Sporik R, Holgate ST, Platts-Mills TA, and Cogswell JJ, (1990), Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. A prospective study, *The New England Journal of Medicine*, 323(9): 502-507.

Sunyer J, Jarvis D, Pekkanen J, Chinn S, Janson C, Leynaert B, Luczynska C, Garcia-Esteban R, Burney P, Anto JM; European Community Respiratory Health Survey Study Group, (2004), Geographic variations in the effect of atopy on asthma in the European Community Respiratory Health Study, *Journal of Allergy and Clinical Immunology*, 114(5): 1033-1039.

Torrent M, Sunyer J, Muñoz L, Cullinan P, Iturriaga MV, Figueroa C, Vall O, Taylor AN and Anto JM, (2006), Early-life domestic aeroallergen exposure and IgE sensitization at age 4 years, *Journal of Allergy and Clinical Immunology*, 118(3): 742-748.

Appendix to Chapter 9

A.9: Sensitivity Analysis, Further Graphs



A.9.1

Sensitivity of the MPI model to +/- 1-% changes in temperature and RH

Following is a number of graphs illustrating the sensitivity of the Popmite model to changes in input variables. It may be helpful to illustrate one of the graphs. For example, in Figure A.9.2 the parameters which have been independently assessed for the sensitivity analysis are on the x-axis (abbreviations for each parameter are described in Table 9.4.1, chapter 9). Each parameter is separated by dashed vertical lines. The Popmite predictions are given on the y-axis, representing the predicted final *adult* population. Each data series represents one hygrothermal input base-case (as summarised in Table 9.4.2, chapter 9). For example, the green squares correspond to the "Average" input hygrothermal base-case. The green horizontal line corresponds to the base-case prediction (nearly 50 mites). The green triangles above and below this line represent the variation in predictions, due to changes in the x-axis input parameters (+/- 10%). For example, a change in input temperature (first variable on the x-axis) results in a *reduction* in predictions (both triangles *below* the solid green line), regardless of whether such change was an increase or a decrease from base-case temperature. On the other hand, changes in other variables do not result in changes in predictions (e.g. input variable "Fast Up"). Figure A.9.3 and A.9.4 are similar to Figure A.9.2, except that they show Popmite predictions for juveniles and for eggs, respectively.

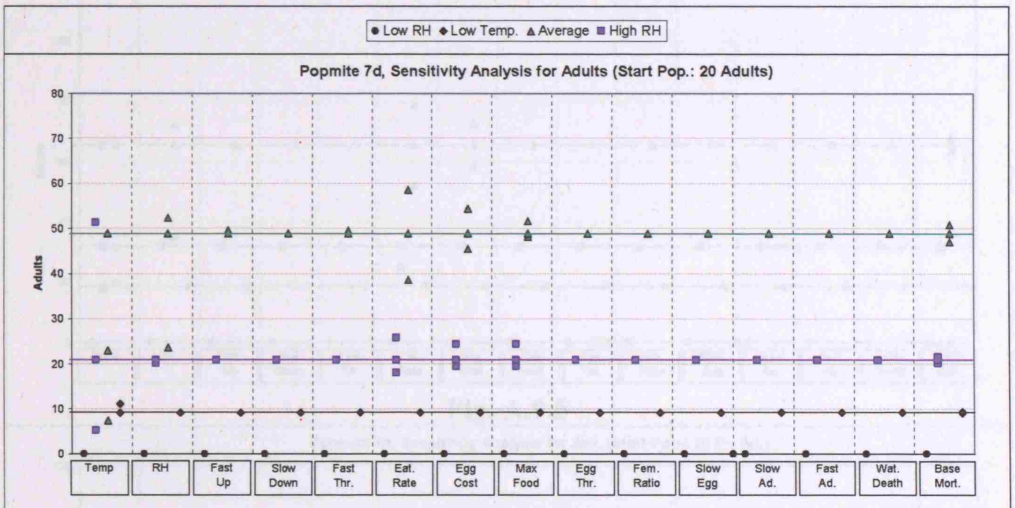


Fig A.9.2

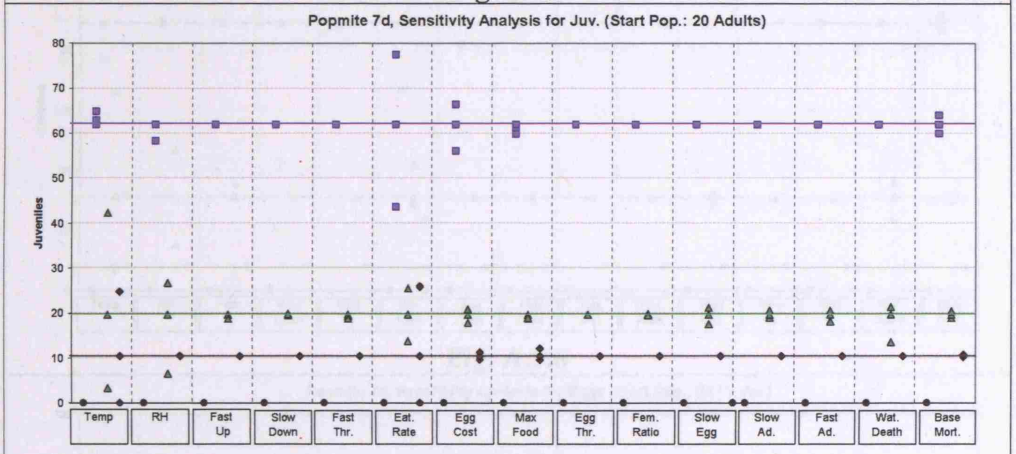


Fig A.9.3

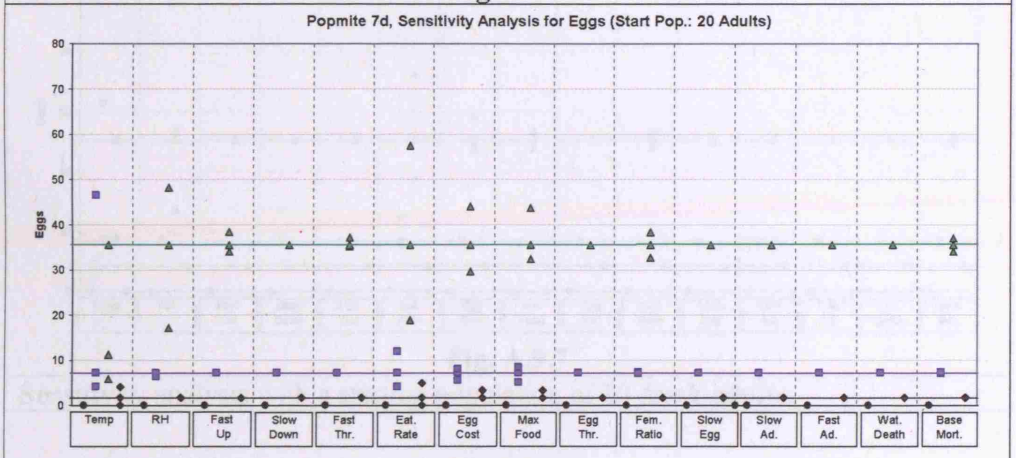
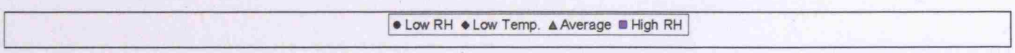


Fig A.9.4

Sensitivity analysis for Popmite, with a starting population of 20 adults.



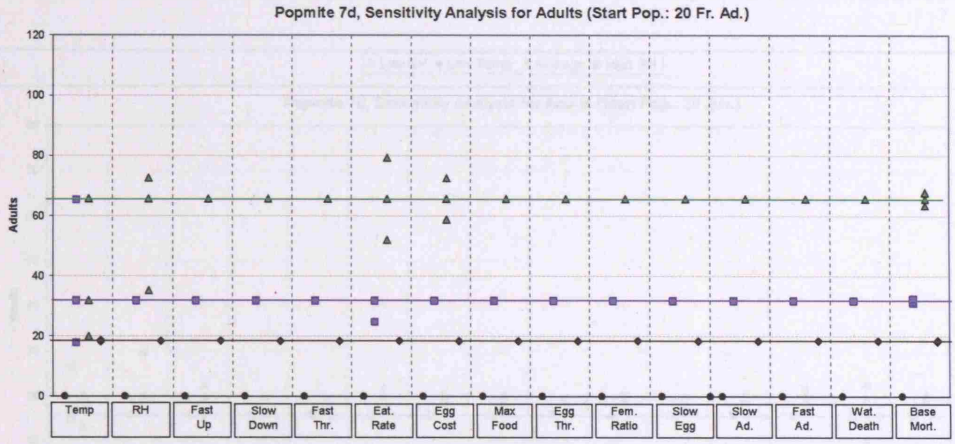


Fig. A.9.5

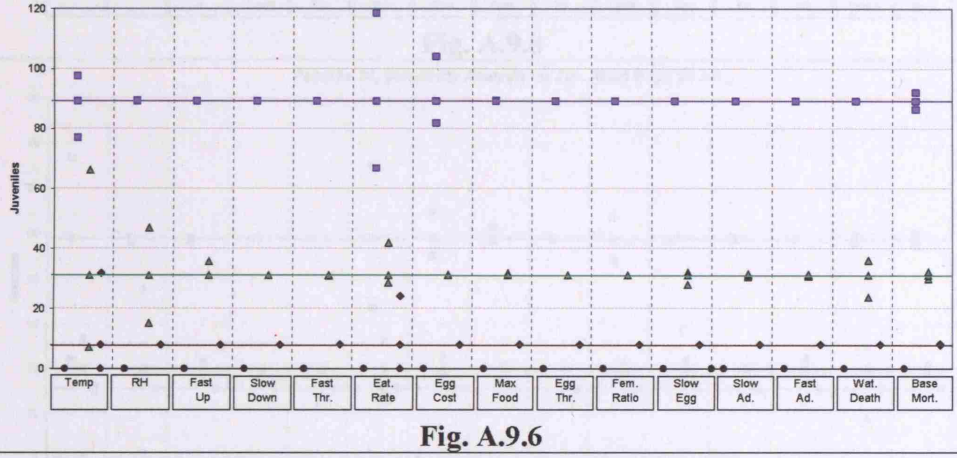


Fig. A.9.6

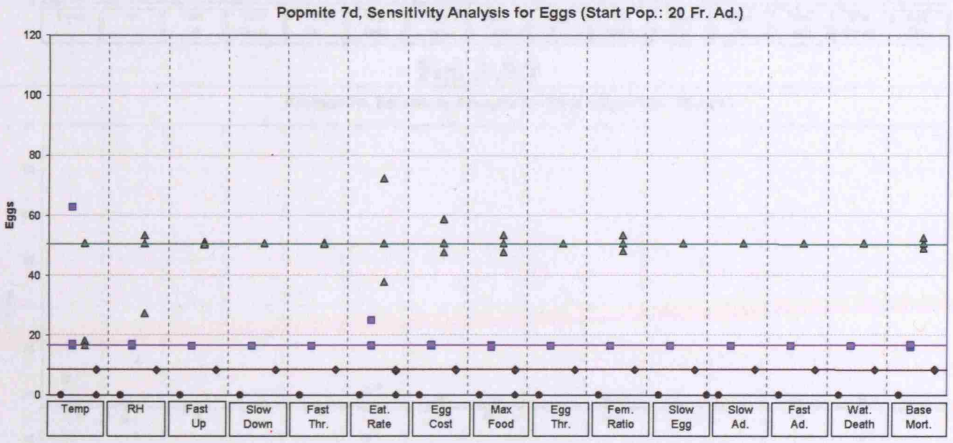


Fig. A.9.7

Sensitivity analysis with a starting population of **20 *fresh* adults.**

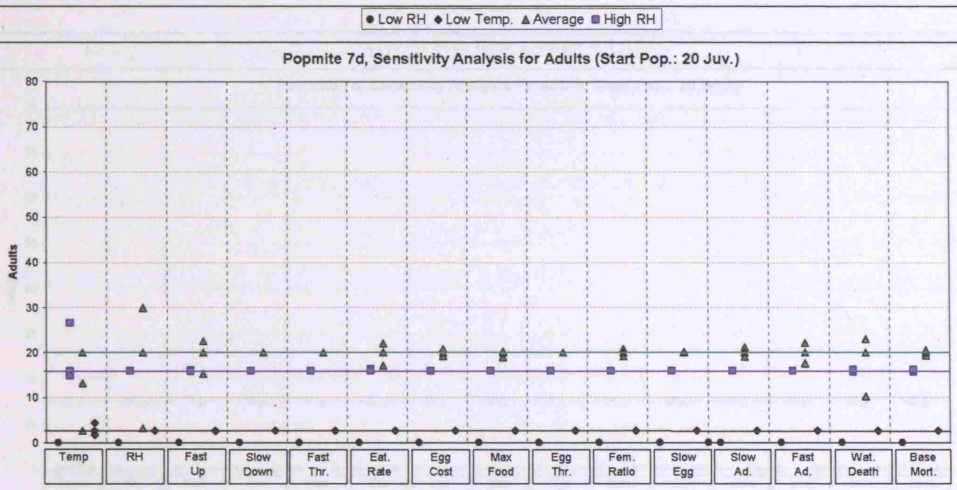


Fig. A.9.8

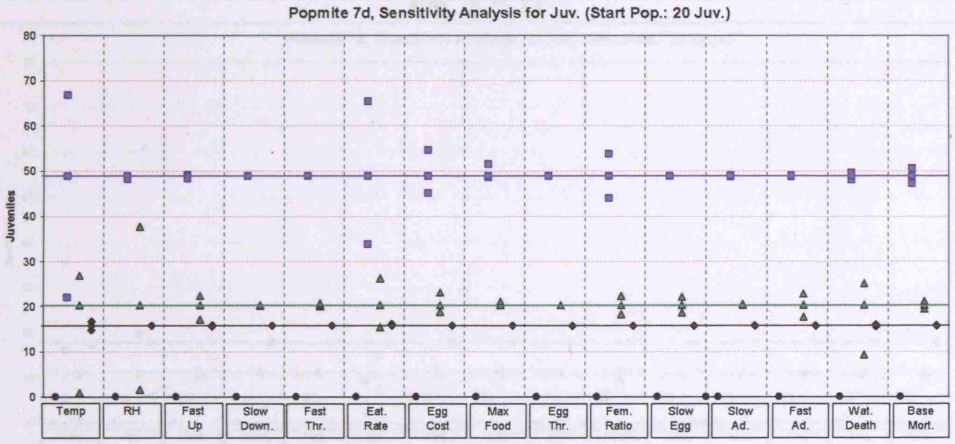


Fig. A.9.9

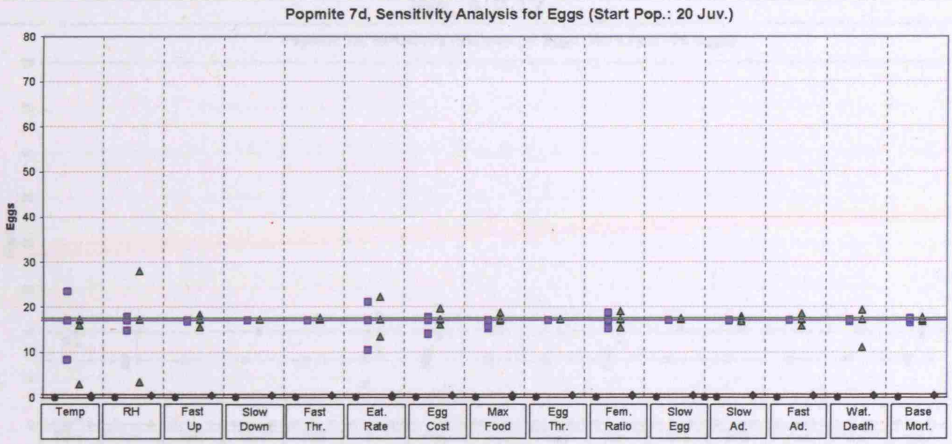


Fig. A.9.10

Sensitivity analysis with a starting population of **20 juveniles** (spread of all ages).

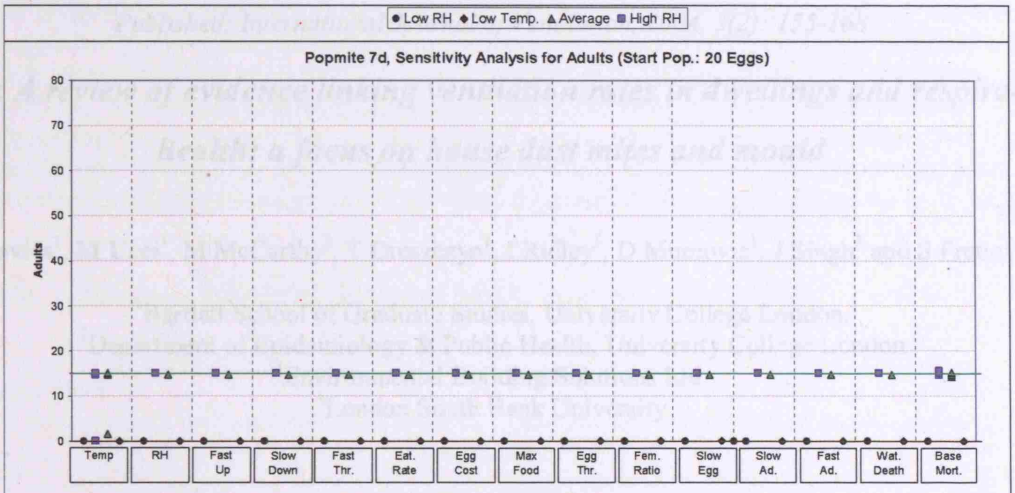


Fig. A.9.11

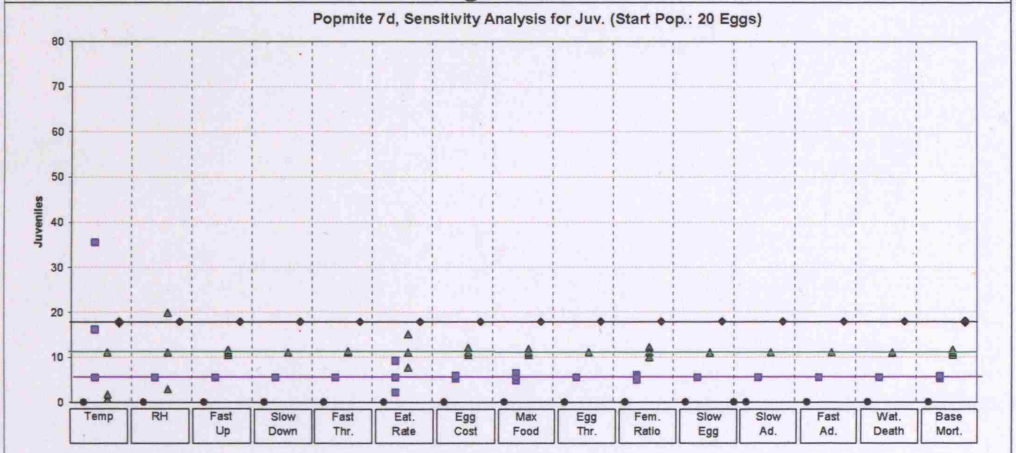


Fig. A.9.12

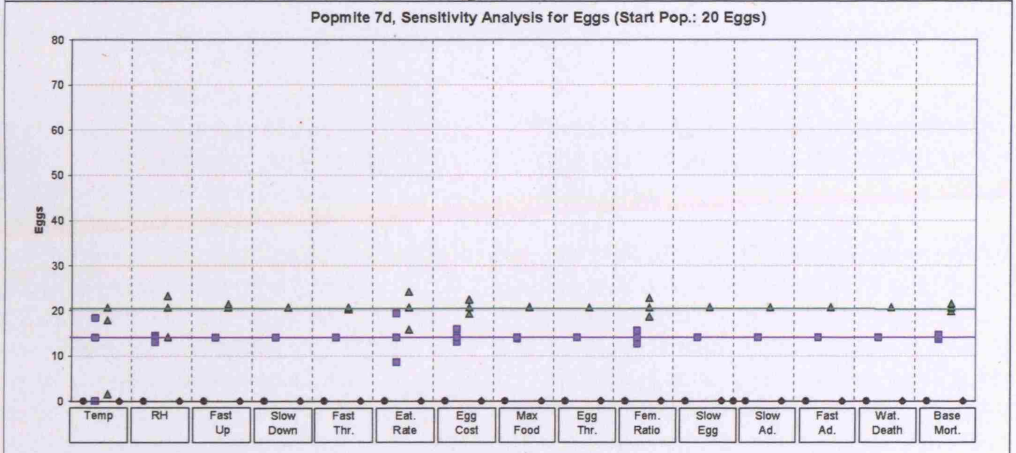


Fig. A.9.13

Sensitivity analysis with a starting population of **20 eggs** (spread of all ages).

Appendix A.0: Published Papers

Published: International Journal of Ventilation, 2004, 3(2): 155-168

A.0.1: *A review of evidence linking ventilation rates in dwellings and respiratory health: a focus on house dust mites and mould*

M Davies[1], **M Ucci**[1], M McCarthy[2], T Oreszczyn[1], I Ridley[1], D Mumovic[1], J Singh[3] and S Pretlove[4]

[1]Bartlett School of Graduate Studies, University College London.
[2]Department of Epidemiology & Public Health, University College London
[3]Environmental Building Solutions Ltd.
[4]London South Bank University

Abstract

This paper reviews the literature for evidence of links between ventilation rates in dwellings and moisture related respiratory health with a particular focus on house dust mites (HDM) and fungal growth. There is general consensus that a link exists between ventilation rates in dwellings and respiratory hazards (for example HDM). There is also general consensus of a link between these respiratory hazards and respiratory problems, but it is not clear to what extent hazards cause ill-health. Most existing data are inadequate for conclusions to be drawn whether ventilation rates directly cause respiratory problems. We discuss the many difficulties in attempting to establish these relationships, and suggest the need for larger studies.

Key words: review, ventilation rates, respiratory health, house dust mites, mould.

1. Introduction

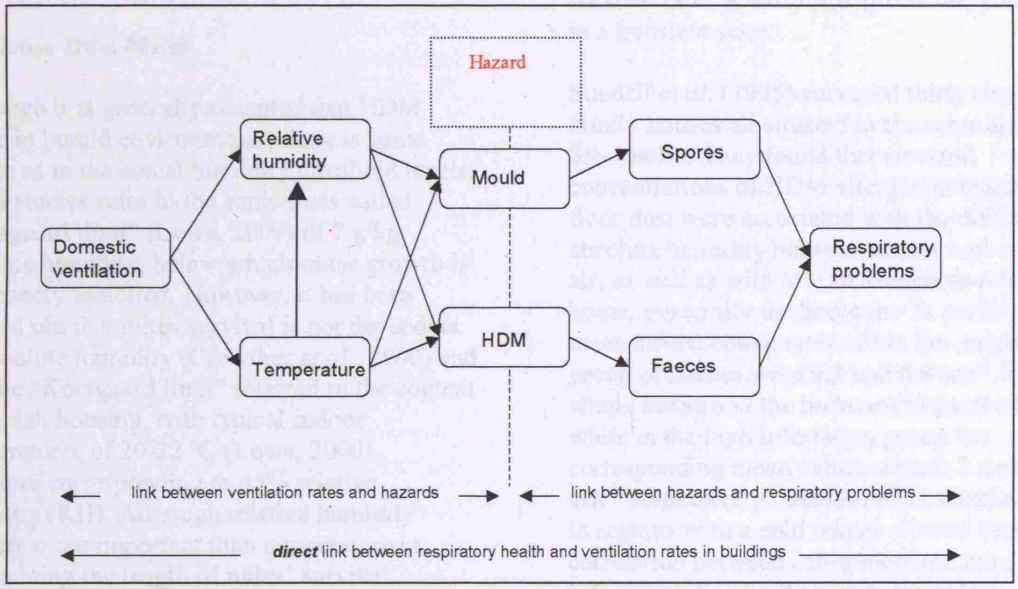


Figure 1. Postulated pathways between ventilation and related respiratory problems

This review focuses on *moisture related* respiratory hazards (in this case HDM and fungal growth). In a most basic manner, figure 1 shows the postulated pathways between ventilation and relevant moisture related respiratory problems (note that this diagram only shows a small element of the total complex system). Convenient sub-categories of links are also shown. The review will deal with these links in terms of publications which relate to:

- a link between ventilation rates in dwellings and moisture related respiratory hazards
- a link between moisture related respiratory hazards and respiratory problems
- a *direct* link between moisture related respiratory health and ventilation rates in buildings

The following three sections examine the evidence currently available in the literature for these links.

Part of the complexity involved in research in this area is that in most cases it is difficult to measure ventilation rates in a meaningful and accurate manner for the number of properties that are required to provide any statistically significant health data. Also, most changes to ventilation rate have occurred at the same time as other changes e.g. other improvements to the building fabric. Frustratingly, it is not easy to infer the ventilation rate of a property from other building factors such as the age of a property or occurrence of draught stripping. In addition, there are theoretical mechanisms which mean

that increased air ventilation is not always beneficial to health (e.g. indoor generated pollutants may decrease but externally generated pollutants may increase).

2. Studies relating to a link between ventilation rates in dwellings and moisture related respiratory hazards

There are two key moisture related respiratory hazards – house dust mites and mould growth. The following two subsections review the environmental conditions required for house dust mites and mould and the link with ventilation.

2.1 House Dust Mites

Although it is generally accepted that HDM thrive in humid environments, there is some debate as to the actual humidity threshold levels. Many studies refer to the sometimes called "Korsgaard limit" (Lowe, 2000) of 7 g/kg absolute humidity, below which mites growth is supposedly inhibited. However, it has been pointed out that mites survival is not dependent on absolute humidity (Crowther *et al.,* 2000) and that the "Korsgaard limit" referred to the context of Danish housing, with typical indoor temperatures of 20-22 °C (Lowe, 2000), therefore corresponding to 45% relative humidity (RH). Although relative humidity appears more important than temperature in determining the length of mites' survival (Crowther *et al,* 2000), the Critical Equilibrium Humidity (CEH) – below which mites dehydration occurs – appears to be dependent on temperature as well (Cunningham, 1996; Arlian and Platts-Mills, 2001). In establishing a limit for the psychrometric control of the mite species *D. farina* Cunningham (1996) suggested that indoor relative humidity should be kept below 40% at 16 °C, 45% at 21 °C and 50% at 26 °C. On the other hand, Raw (2001) states that RH at 35-40% in winter, although difficult to achieve in the UK, should be adequate to prevent mite proliferation. Temperature is also an important factor for the egg to adult time span.

It is important to note that HDM survival is not only dependent upon microclimatic hygrothermal conditions but also upon the length of time for which such hygrothermal conditions occur. For example, some studies suggest that adult mites die of dehydration in 5 to 11 days, depending on temperature (25 °C - 34 °C) when continuously exposed to RHs of 40% or 50% (Arlian and Platts-Mills, 2001). It should also be pointed out that mites are able to move to areas where more favourable environmental conditions occur. Furthermore, some differences have been found between laboratory and 'wild' HDM populations (Crowther *et al.*, 2000). However, most research studies on mites' survival ability have been conducted under steady-state laboratory conditions using cultured populations of HDMs. Although much has been established on HDMs' physiology, further research is required providing a complete knowledge on wild mites' survival rates at various hygrothemal conditions in a transient state.

Sundell *et al.* (1995) surveyed thirty single-family houses all situated in the same area of Stockholm. They found that elevated concentrations of HDM allergen in mattress and floor dust were associated with the difference in absolute humidity between indoor and outdoor air, as well as with low air-exchange-rates of the home, especially the bedroom. In particular, the mean air-exchange-rates of the low infestation group of houses were 0.3 and 0.9 ach^{-1} for the whole house and the bedroom respectively, while in the high infestation group the corresponding mean values were 0.2 and 0.2 ach^{-1} respectively. Sundell *et al.* concluded that in regions with a cold winter climate there is a correlation between infiltration and mite infestation, but air-flow rates related to number of people in the home appears a stronger indicator of HDM infestation than air-flow rates related to home volumes.

Several studies have been carried out where mechanical ventilation with heat recovery (MVHR) and/or dehumidifiers were utilised in order to reduce relative indoor humidity levels in winter and consequently mite concentrations. However, although the use of MVHR appears to have proven quite successful in Scandinavian countries (e.g.: Emenius *et al.*, 1998; Harving *et al.*, 1994), such an approach has caused some conflicting results in the UK. A study (Fletcher *et al.*, 1996) conducted in the North-West of England on 18 houses – 9 with MVHR and 9 control houses – concluded that "the MVHR unit does not reduce indoor humidity to levels capable of retarding mite population growth and decreasing mite allergens in the type of houses

predominantly found in the mild and humid climate of the North-West of England" (*ibid.*, p.1051).

In a later study (Niven *et al.*, 1999), an additional central dehumidification modification of the MVHR (MVHRcd) was adopted in order to further assess the viability of the psychrometric control of HDM in the UK. Ten active houses were fitted with adapted MVHRcd units; relative humidity and allergen levels were monitored for 15 months and compared with the correspondent results of 10 control houses. The target temperature and humidity for the bedrooms in the active houses were 45% RH or 7 g/kg absolute humidity at 21 °C. The researchers concluded that "the MVHRcd system failed to confer a benefit in terms of mite allergen reduction" (*ibid.*, p.756). However, the authors also pointed out that the buildings' air-tightness might have compromised the effectiveness of the MVHRcd system. No measurements of air-infiltration were carried out to determine is this was a possible explanantion.

In a study carried out by Berry *et al* (1996), it was shown that the houses which rarely or never opened windows had higher mean yearly average mite counts taken from the bedroom carpet. This was not observed with counts obtained from the living room carpet, when the degree of opening the living room window was examined.

Several studies report that HDM numbers increased with the severity of the dampness in the property (Adan *et al* 1988 and Hart and Whitehead 1990). Toma *et al* (1993) found that ventilation did not influence mite numbers, but Korsgaard (1979) found that there was a tendency for higher numbers of mites in houses where the number of airing hours (opening up windows) was low. Korsgaard (1979) also found that there was no correlation between the temperature and mite counts. However, Irie *et al* 1990 found a link between increasing mite numbers and increasing indoor temperatures.

Modeling can provide an insight into this issue. As a result of a two-year research project funded by EPSRC in the UK, two models have been developed for the prediction of HDM in houses (Crowther *et al.*, 2002). The model 'BED3' has a steady-state hygrothermal model linked to an empirical population model. For the latter, laboratory measurements were undertaken with populations of HDM kept for three weeks at different combinations of steady RH and temperature, covering the range of conditions typical of UK dwellings. The more complex model 'Lectus' is a transient, three-dimensional model that simulates all stages of mite development. The population model adopted in Lectus is based on published data for the *Dermatophagoides pteronyssinus*, the most common mite species in the UK. With regard to the Lectus population model, the authors of the research pointed out that the existing published data are not complete but there was sufficient information, making simple assumptions, to derive curve-fitted equations. The models BED3 and Lectus were used to examine a range of issues, including the effect of ventilation rates on HDM populations. The study concluded that:

- Small reductions in ventilation rate below 0.5 ach^{-1} can have a dramatic impact. Modelling suggests that reducing from 0.5 to 0.4 ach^{-1} can increase the mite population by 100 times. However, an increase to above 0.7 ach^{-1} can also lead to an increase in the mite population in a fuel poor dwelling
- Raising bedroom temperatures from 16°C to 18°C, i.e. without reducing ventilation, can result in a significant (factor of ten) reduction in mite numbers. The increase in bedroom temperatures over the last 50 years, partly as a result of increased central heating and improved insulation, is therefore likely to have had beneficial effects. This supports the case for continuing to improve the UK's housing stock.
- Modelling suggests that building occupant density is a key parameter in determining house dust mite populations. Increasing the number of occupants in a dwelling from 4 to 6 can increase the mite population by 10,000 due to the increased moisture production in the property.

The role of indoor temperatures in the correlation between air-leakage and HDM population is also highlighted by Lowe (2000). The author considered the Critical Equilibrium Humidity (CEH) defined by Cunningham (Cunningham, 1996) of 40% RH at 16 °C and 45% at 21 °C. Modelling based on a typical UK dwelling and Kew weather data showed that at low internal temperatures the "Cunningham limit" is exceeded for most of the winter and increasing the ventilation rate does not improve

the situation greatly. At high internal temperatures, problems appear likely to occur only at ventilation rates significantly less than 0.5 ach^{-1}. Ridley *et al.* (2003) also found through modelling that an air-infiltration rate below 0.5 ach leads to an indoor RH greater than 70% in fuel rich dwellings (0.7 ach^{-1} for fuel poor dwellings). While in fuel rich dwellings the RH rapidly decreases with an increase in air-leakage, in fuel poor dwellings (who cannot afford to maintain always comfortable indoor temperatures) such inverse correlation does not occur.

2.2 Mould

Woolliscroft (1997) stated that the high level of condensation and mould in the UK is the consequence of the small size of the dwellings, low temperatures, high absolute humidity of the incoming air, and high occupancy of dwellings. 35% of dwellings were affected by condensation and 17% by mould growth. Comparing these results which were based on the English House Condition Survey 1988, with the same survey in 1996, it can be noticed that the incidence of mould growth of any severity has fallen to 14.6% of the total stock (DETR, 1996). The latest house condition survey published in 2001 omitted the collection of any condensation and mould data (ODPM, 2003).

A study by the UK Building Research Establishment (BRE) (Research project number EP228, 1990) revealed that recently built one bedroom and bedsit homes in the UK had significant condensation problems, which could lead to mould growth and proliferation. This study gave an indication of the factors related to condensation for example ventilation, air movement, heating and insulation. Their study indicated that the factors such as ventilation, air movement, heating and insulation were more important than occupant behaviour and energy consciousness and the most important occupant characteristics were the number and age of occupants (Raw and Fox 1990 in BRE study, Research project number EP228). BRE (Hunter *et al* 1988 and 1996) carried out biological assessments of houses and their investigation revealed that the most influential factor affecting the fungal counts appeared to be season.

In summary, it would appear that there is general consensus that links do exist between ventilation rates and moisture related respiratory hazards. We will now move on to consider the links between these hazards and possible respiratory problems.

3. Studies relating to a link between moisture related respiratory hazards and respiratory problems.

The most commonly perceived health effect arising from exposure to airborne moulds and other microorganisms, for example HDM, is allergy. Allergy-related diseases include asthma, rhinitis, and eczema or the less common diseases of extrinsic allergic alveolitis (hypersensitivity pneumonitis) and allergic bronchopulmonary aspergillosis (Pope *et al* 1993). Again, this section initially discusses the link between mites and health before moving on to consider mould.

3.1 House Dust Mites

HDM allergens are mostly present in their faecal pellets and they can trigger Type I allergic reactions, including asthma. Some studies also suggest that HDM allergens are associated with other health problems such as eczema and perennial allergic rhinitis. The evidence is reviewed by Raw, (Raw, 2001) who reports that "levels of mite allergen in the dust in most UK homes are high enough to cause sensitisation and it is possible that most people in the UK are exposed to enough mite allergen to cause asthma if they are susceptible to this disease for genetic or other reasons" (*ibid.*, 2001, p.15). However, the contribution of HDM as a direct cause of asthma, in comparison with many other predisposing and precipitating causes, is not known

Generally, the effects of pollutants on the lung can be categorised as irritation, inflammation, bronchoconstriction and sensitisation. Some of the more potent agents of allergic lung disease are found in indoor environments; such aeroallergens (house dust mite and moulds) have been recognised for many years. Inner city children have the highest prevalence and the highest mortality rates for asthma in the USA (Call *et al* 1992) and these children also have a high prevalence of dust mite sensitisation (Platts-Mills and Weck, 1989). However, many other factors could also contribute to this relationship

House dust mites, moulds and, less commonly, amoebae can colonise building structures, services, furnishing and finishes (e.g. Singh, 1999). House dust mites, fungi and yeasts are potent sensitizers, and they flourish in an environment of high relative humidity and low ventilation. Fragments of these organisms or their decayed material or their metabolites, becoming airborne, can be inhaled and cause allergic disease.

An important meta analysis of 23 patient-level intervention studies (Goetzsche *et al* 1998) was derived from 229 reviewed papers. A total of 230 patients, all showing mite sensitivity shown by skin-prick, were divided between intervention cases and non-intervention controls. Interventions were: 6 using chemical methods, 13 physical methods and 4 combination. No statistical difference was found in symptom responses between intervention and controls. The authors consider the sample size of strong statistical power and so the 'most likely explanation' given is either insufficient reduction in house mite levels or other allergens coexisting. In a dissenting editorial, Strachan (1998) suggested that sub-group analysis could still reveal clinically useful interventions with larger studies.

3.2 Mould

Now turning to mould in more detail, a number of studies have shown that mould growth in damp housing was associated with childhood respiratory illness; wheezing and asthma (Su *et al* 1990; Flannigan *et al* 1990; Dales *et al* 1990; White 1990; Spengler et al 1993; Husman *et al* 1993). None of these studies was in a peer-review health journal, and do not necessarily demonstrate a causal relationship

The effect of damp and mould in the home on respiratory health was reviewed by Peat *et. al.* (1998). The reviewer suggested that houses need to be specifically designed for primary prevention of respiratory problems associated with indoor allergen proliferation rather than using post hoc procedures to improve indoor climate and reduce allergen load as a secondary or tertiary preventive strategy. It was strongly emphasised that studies with large sample sizes were needed to measure whether intermittent peak exposures or low cumulative exposures to indoor allergen pose a clinically important risk.

Mould in damp buildings has recently emerged as an indoor environmental hazard of some concern (Lange et al 1993, Singh 1994a and Rylander 2003), although the issue has existed for centuries (Rautuiala *et al* 1998). A large volume of literature is appearing in journals particularly related to characteristics, distribution, public health, exposure, and health relationships for microbes, including fungi, and the indoor environment (Kalliokoski 2003, Lugauskas *et al* 2003, Adeeb and Shooter 2003,Sarca *et al* 2002, Menetrez *et al* 2002, Menetrez and Forde 2002, Kemp *et al* 2002a, Kemp *et al* 2002b and Mussalo-Rauhamaa *et al* 2003).

There are more than 100,000 species of fungi, and the genera and species possibly linked to human disease involve a wide array of both common and rare moulds. Fungi produce large numbers of spores and when these spores are liberated from infected buildings to the indoor air, they can be regarded as organic dust. These spores can, like other types of dust, sediment on surfaces or can be inhaled by occupants and deposited on the mucosal surface of the upper airways and in the eyes.

Microorganisms and their metabolites may cause a range of respiratory symptoms, depending upon the species, the exposure and the immune status of the subject (Singh, 1994a; Singh 1994b; Lacey, 1994; Comtois and Garcia, 1994).

Garret *et al* (1998) reported that no significant association between total viable mould concentrations and health outcomes was seen despite significant associations with specific genera. Reporting on the findings of the PEACE study, Andriessen *et al* (1998) concluded that Peak Flow (PEF) variability in atopic children was associated with (but not necessarily caused by) reported moulds in the home.

A few governmental agencies have published guidelines on mould assessment and remediation but most are very general in nature (Minnesota Department of Health. 2001and New York City Department of Health 2000 and Rao *et al* 1996). Some guidelines focus on toxogenic moulds, including Stachybotrys chartarum, which have

been reported in association with health conditions including acute pulmonary haemorrhage (Chapman 2003).

It is helpful to conclude this section of the review with the findings of a recent report (Raw *et al,* 2001). This study placed HDM in the highest level risk group with regard to health and safety hazards in homes. Fungal growth was placed in the second highest level risk group. In this study, hazards were placed in rank order, from the perspective of deciding whether preventative action was needed. It appears that the actual health risk from mould in buildings has yet to be quantified e.g. Chapman *et al* (2003) and Bornehag *et al* (2004). While repeated exposure to large amount of fungal propagules risks the development of specific allergic reactions, there is no adequate evidence of serious health hazards caused by so-called 'toxic' moulds.

In summary, there appears to be general consensus that a link exists between HDM and mould (i.e. respiratory hazards) and respiratory problems Having previously indicated that there is also general consensus that links do exist between ventilation rates and these respiratory hazards we now conclude by exploring what evidence exists to support a *direct* link between ventilation rates and respiratory problems.

4. Studies relating to a *direct* link between moisture related respiratory health and ventilation rates

The EUROVEN group recently reviewed the scientific literature relating to the effects of ventilation on health, comfort and productivity in non-industrial environments (Wargocki *et al* 2002). The study concluded that there was a strong association between ventilation and health. Studies judged conclusive implied that low ventilation rates in homes may be one of the factors exacerbating allergies due to the increased rate of infestation of HDM. The study also noted however, that more information is required on links between ventilation rates and health in homes.

A recent study (Emenius *et al* 2004) was undertaken to examine the impact of building characteristics and indoor air quality on recurrent wheezing in infants. The study found that whilst building-related exposures appear to have a major impact on children's health, this was not primarily explained by differences in ventilation systems, air change rate or HDM infestation.

In some UK studies the adoption of MVHR appeared successful for HDM control (Howieson *et al.,* 2002; Htut *et al.,* 1996; and McIntyre, 1992). However, not all of the studies measured ventilation rates nor the clinical efficacy of the remedial measures.

Howieson *et al.* (2002; 2003) examined the effect of a number of remedial measures (including MVHR, steam cleaning, new bedding) on 54 asthmatic subjects in North Lanarkshire. The study concluded that lung function measurements and health questionnaire data confirmed a significant improvement in the active group compared with the control group. However, the study presented a number of confounding variables. For example, no pressure tests were carried out. In addition, no skin prick tests were undertaken and consequently the project could not differentiate between the health effects influenced by a reduction in HDM allergen levels and/or the overall improvement on indoor air quality produced by greater ventilation rates.

In another UK research project adopting MVHR, twenty houses in the Southampton area were fitted with MVHR and a further 20 houses acted as controls (Stephen *et al.,* 1997; Warner *et al,* 2000). As air-infiltration was also measured, the effect of air-leakage on indoor humidity was explored using linear regression. It emerged that the houses group with MVHR showed a significant effect of leakiness with leaky houses having lower humidity. In the control houses, however, it appears that humidity was not affected by the houses' leakiness. However, the independent analysis of the two groups does not show that the two results are statistically different from each other. As regards the effect of MVHR on allergen concentration, Warner *et al* (2000) noted that there was evidence for a beneficial effect of MVHR on *Der p1* levels. However, the reduction in allergen levels did not result in a significant clinical improvement - the power of this study was low to detect clinical changes. "The likelihood of being able to show a change in clinical symptoms could be improved by performing a larger study and ventilating more areas of the houses, possibly with the inclusion

of active dehumidification within the systems." (*ibid*).

Many studies do not attempt to investigate the links between ventilation and health but rather between *dampness* and health. Ventilation and damp can be closely related and so such studies are reported here. There is however, a lack of clarity in the literature as to the definition of a 'damp' building. For example, high absolute humidity, high relative humidity, high moisture content of elements of the fabric and the occurrence of mould are all possible indicators. This definition is critically important because whereas, for example, the absolute humidity will always tend to drop with increased ventilation (unless the external absolute humidity is higher), relative humidity is a function of both temperature and the moisture content of the air and so increased ventilation can, under some circumstances, increase relative humidity.

A recent report (ISBE, 2003) concluded that extensive knowledge exists on the influence of humid environments to human health. Health problems in damp and humid buildings were first described by Leeuwen (1924) and these problems related to buildings have received increased attention over the last few decades. In the last two decades several large studies in the UK, USA, Scandinavian Countries and the Netherlands strengthened confidence in the relationship between indoor environmental humid conditions and an increase in asthma, impaired respiratory function, general respiratory symptoms and respiratory infection among children (Brunekreef *et al* 1993; Dales *et al* 1991; Cuijpers *et al* 1995; Li and Hsu 1996 and Rylander 2003)).

A recent review of the literature on dampness and HDM exposure in buildings and health effects (Bornehag *et al* 2004) concluded that dampness is a risk factor for health in domestic environments but that the literature is not conclusive in respect of causative agents. The strong need for more multidisciplinary studies was noted.

A recent study (Hagerhed *et. al.,* 2002) reported that dampness (inferred from visible signs of mould and condensation, coupled with the perception of indoor air quality e.g. 'stuffy air') is more common in older buildings and buildings with natural ventilation. Bornehag (2002) summarised 15 different studies on dampness and health concluding that in 13 studies a positive association was found between dampness and health effects namely asthma and wheezing.

A study conducted in Scotland (Williamson, 1997) showed that asthmatic patients attending a hospital asthma clinic were two to three times more likely to live in a dwelling with evidence of dampness (inferred from fabric moisture content and severity of mould) than an age and sex matched random sample of the general population living in the same area of the city of Glasgow.

Further confirmation of the significant influence of house dampness and mould on health status is reported in numerous, mainly medical, reports and journals (Garrett *et. al.*, 1998; Andriessen, *et. al.*, 1998; Zock, *et. al.*, 2002; Zureik, *et. al.*, 2002).

As noted earlier, some of the studies reported here do not deal directly with ventilation – rather 'damp' housing. The two are related but it is clear that the number of studies which have attempted to link ventilation rates directly to respiratory problems are scarce.

It may be helpful to conclude this section with the findings of the National Academy of Sciences, USA. The results of workshops in 1999 (Committee, 2000) reported 'existing data are inadequate for conclusions regarding the association between ventilation rates or ventilation system microbiological contamination and either the exacerbation of asthma symptoms or asthma development.' 'Airtight building envelopes and low rates of ventilation have been cited as factors that may contribute to asthma incidence or symptoms or may explain recent increases in asthma; however, very few relevant data are available ... .measurements of ventilation rates should be included, when possible, in future asthma case-control studies or cross-sectional surveys.'

5. Discussion

The literature review highlights the limited research that has been undertaken to demonstrate a *direct* relationship between domestic ventilation and health. The medical and building science literatures include many publications addressing ventilation and health in

offices, but few domestic housing studies. There have been studies by occupational health services to support the growing white-collar workforce but because private housing is not regulated by health and safety legislation there are fewer relevant studies. Also, it is relatively difficult to measure ventilation in housing: current techniques can only be undertaken on a small number of dwellings at a time. Yet, health studies generally require large occupant samples.

Health effects of pollutants can be studied in three ways – medicine, toxicology and epidemiology. Typically, the first is of concern in relation to an individual person. The person has unexpected symptoms, and explanations for these are sought in the surrounding environment. If a particular aspect of the environment is suspected, then two further directions are possible: toxicological studies will test the possible agent in laboratory settings (for example, effects on tissue cultures or mice); epidemiological studies will compare the frequency of the disease in people exposed to the pollutant with people not exposed.

This approach has served industrial medicine well, and has identified potential hazards of particular working environments, for example dyes in the chemical industry causing bladder cancer and asbestos in the construction industry causing lung mesothelioma. But assessing the effects of pollutants in the home is more complicated. People living at home have a varied environmental exposure (living in different rooms, working with different materials, periods of time in and out of the house) and these are not usually recorded before the onset of symptoms. The levels of exposure are probably below industrial levels, and health records not so accessible. In general, whereas occupational health services assess the health of workers in the workplace, general medical services rarely see people in their homes except for nursing and care services.

In the industrial examples given above, the diseases identified were relatively unusual. On the other hand, respiratory symptoms that may be attributed to housing are widely prevalent. Coughs and colds affect most people every year. Moderate or severe symptoms can arise from infections (bronchitis and pneumonia) and 'asthma', (a state of narrowing of respiratory airways). The United Kingdom has the largest proportion of people in Europe who believe they have asthma – one in three of the population by current surveys. (Whether the country comparisons are accurate is another question: the rapid growth in self-diagnosis of asthma suggests a possible re-allocation of other respiratory complaints {coughs and colds}). Respiratory symptoms are common; and so also are the various factors suggested to cause them. To focus on asthma again, the range of possible 'causes' include respiratory infections, chemical vapours in the air, ingested chemicals, dust-borne particles, temperature changes, smoking, exercise and psychological reactions. Asthma symptoms occur because of inflammation of the respiratory tract. 'Allergy' is only one of several pathways for the body to create the inflammation, and people who are 'allergic' don't necessarily have that as the cause of specific asthma symptoms – which may be the result of a simple viral infection (cold, cough) or increased obesity making exercise more difficult.

Good studies relating home air to respiratory symptoms are difficult to achieve. Symptoms are common and difficult to standardise, while exposures are equally hard to record. Typically, studies have tried to focus on asthma as a disease, since it occurs in children and there appear to be a wide variety of triggers. Three sorts of epidemiological studies can be used: Cross-sectional studies are most common. These surveys record existing exposures in housing and existing levels of disease, and look for statistical associations between the two. The weakness in these studies is 'confounding' – that is, several factors may be identified statistically, but another factor (perhaps unrecorded) may be having a greater real effect. Other criteria, including strength of association, biological plausibility and specificity of the effect should be considered in evaluating the results.

Case-control studies compare people with symptoms against people without symptoms, and look for different levels of exposure. These studies can look at past exposure, and therefore show whether exposure happened before disease – which cross-sectional studies cannot. Nevertheless, it is necessary to collect information on all possible causes, and still the case-control design does not exclude confounding factors.

Longitudinal population studies have the strongest scientific design. They follow a cohort of people forwards in time. They measure the exposure before onset of the disease, and therefore help avoid confounding factors. However, they have to be large, since only a proportion of people tracked over time will develop the symptoms. This in turn means measuring ventilation over a large number of properties which is very expensive.
In the field of domestic ventilation and health so far, only two sides of the equation have been assessed in detail: there have been studies of the effects of ventilation on air characteristics, such as dust mites, mould spores, carbon monoxide; and there have been studies showing how these factors are associated with respiratory symptoms and diseases. But, in contrast to office studies, there have been relatively few *direct* studies of domestic ventilation and diseases.

It may be reasonable to accept the separation of the 'technical' studies of ventilation from the 'medical' studies of air and disease. Thus, if ventilation reduces pollutants, then potential harm may be reduced. But it would be welcome to know whether there was a real effect because the implications of ventilation are not negligible. Energy efficiency seeks to maintain indoor temperatures while reducing heat loss. 'Tight' buildings have reduced levels of natural ventilation; but they may have increased pollutants, for example, high concentrations of domestic chemicals (e.g. cleaning materials), cooking gases (carbon monoxide) or house dust mites (through higher humidity). These in turn are believed to have respiratory symptom consequences, but the link to levels of ventilation is unknown – making it difficult to give appropriate guidance.

Two other points are relevant for the discussion. First, the concept of 'attributable risk'; this suggests how much a factor contributes to a disease (attributable fraction) or how many people may be affected (population attributable fraction). While the attributable fraction may be low – say only one in twenty cases, if the disease is widespread then the population effect can be quite large in number of cases. (Take, for example, the view that current levels of external air pollution are causing more than 10,000 premature deaths in Britain each year.) A scientific study of ventilation would wish to make some numerical estimate of the health effects.

Second, it is probable that there is a threshold level for some pollutants. Equally, it is possible for human bodies to excrete or detoxify some chemicals. We do not have adequate knowledge of thresholds and should not assume that only a perfectly 'clean' environment is healthy. Ventilation itself is not a health hazard. However there is considerable theoretical evidence to support the hypothesis that ventilation rate around the levels controlled by the Building Regulations can have a health effect. The pathways are summarised in Figure 1. However, this diagram hides the complexity in trying to identify if a real link exists. Ventilation may impact on hazards and then respiratory problems which are not only affected by spores and faeces but also by other hazards. This is made more complex by the real difficulties in monitoring ventilation, hazards and respiratory problems in one study. It is these difficulties in monitoring that may, in part, explain the often conflicting results from different studies. Being able to monitor ventilation, HDM populations, mould growth and respiratory problems is key to determining a link. The difficulties in monitoring respiratory problems are discussed above but an important issue also relates to most studies relying on self reporting rather than any medical diagnosis.

Measuring the actual hazards (mould and house dust mites) is also problematic. Although the English House Condition Survey has developed a method to rate the severity of mould growth by surveyors, many studies simply rely on the occurrence of any mould, condensation or damp - terms which can have a wide range of different interpretations. It is possible for example to hypothesise a scenario where the occurrence of condensation could *reduce* mould and house dust mites. For example, condensation on single glazing may be a useful stimulus for occupants to increase occupant controlled ventilation which in turn would lead to less mould and HDM. The advantage of monitoring mould is that it is at least visible and so self reporting is possible. However, there is the complexity that mould may be evident due to a previous problem which no longer exists if it has not been cleaned up. In the case of HDM, these are not visible and monitoring is very difficult. HDM populations also vary seasonally due to the change in outside vapour pressure. Sampling is very dependent on the methods used, e.g. vacuum cleaning area of the building,

since mites are invisible there may be a colony of mites which happen to have the correct micro-environment in a location which has not been sampled. Also it is not the mites themselves that cause the respiratory problems but their faeces which have a long life and can be present even if the colony has been eradicated.

As noted earlier, domestic ventilation is also very difficult to measure on the scales required and no reliable methods of inferring domestic ventilation rates have been developed. The most commonly measured parameter to infer ventilation rates is pressure testing. However, pressure testing only reveals something about the background air infiltration and nothing about the occupant controlled ventilation.

6. Conclusions

A key change in this field in recent years has been a move from extrapolating from laboratory studies of air pollution causing respiratory symptoms to the use of a limited number of epidemiological studies of the real world for long-term effects.

An extensive body of literature exists which attempts to investigate relationships between ventilation and indoor air quality. There is general consensus that a link exists between ventilation rates in dwellings and respiratory hazards (for example HDM). There is also general consensus that a link exists between these respiratory hazards and respiratory problems. For relevant moisture related respiratory hazards (HDM and fungal growth), the literature offers some advice on the minimum required ventilation rates to prevent unacceptable hazard and the consequent respiratory health risks.

Of particular interest though, it appears that most existing data are inadequate for conclusions to be drawn regarding the *direct* association between ventilation rates and respiratory problems. It is noted that there are many real difficulties in attempting to establish such a relationship and further work may be required to achieve this

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8. References

Adan, O., Schober, G., Kniest, F. and Vorenkamp, J. (1988) "Modification of indoor humidity conditions: an allergological sanitation method for the indoor environment", *Revue Francaise D'Allergologie et D'Immunologie Clinique*, Vol. 28, pp. 147-151.

Adeeb, F., Shooter, D.(2003) "Emission and evolution of air-borne microflora in slaughter houses", *Indoor-Built Environ,* Vol. 12, pp. 179-184.

Andriessen, J.W., Brunekreef, B., Roemer, W. (1998) "Home dampness and respiratory health status in European children", *Clinical and Experimental Allergy*, Vol. 28, pp. 1191-1200.

Arlian, L.G., Platts-Mills, T.A. (2001) "The biology of dust mites and the remediation of mite allergens in allergic disease", *Journal of Allergy Clinical Immunology*, Vol. 107 (3 Suppl.), pp. S406-13.

Berry, R.W., Brown, V.M., Coward, S.K.D., Crump, D.R., Gavin, M., Grimes, C.P., Higham, D.F., Hull, A.V., Hunter, C.A., Jeffery, I.G., Lea, R.G., Llewellyn, J.W. and Raw, G.J. (1996) "Indoor air quality in homes: part 1. The Building Research Establishment Indoor Environment Study", London, Construction Research Communications.

Bornehag, CG, Sundell, J, Bonini, S, Custovic A, Malmberg P, Matricardi, P, Skerfving

S, Sigsgaard, T & Verhoeff, A. (2004) Dampness in buildings as a risk factor for health effects, EUROEXPO. A multidisciplinary review of the literature (1998–2000) on dampness and mite exposure in buildings and health effects Indoor Air Vol. 14(4) pp. 243 - 257.

Bornehag, C.G. and Sundell, J. (2002) "Dampness in buildings as a risk factor for health effects", in: *Proceedings: Indoor Air 2002. 9th International Conference on Indoor Air Quality and Climate*, Monterey, Indoor Air 2002.

Brunekreef, B., Verhoeff, R.T., van Strien and van Wijnen. (1993) "The role of sensitisation to dust mites and moulds in explaining the relationship between home dampness and childhood respiratory symptoms", in: *Proceedings of Indoor Air 93: 6th international conference on indoor air and climate*, Vol 1, pp 153-158.

Building Research Establishment, (1990), "Research project number EP228", Garston, BRE.

Call, R.S., Smith, F.T., Morris, E. Chapman, M.D., Plat-Mills, T.A.E. (1992) "Risk factor for asthma in the inner city children", *The journal of paediatrics*, Vol 121, pp. 862-866.

Chapman, J.A., Terr, A.I., Jacobsn R.L., Charlesworth, E.N., Bandana, E.J. (2003) "Toxic mould: phantom risk vs. science", *Ann Allergy, Asthma and Immuno.*, Vol. 91, pp. 222-232.

Committee on the Assessment of Asthma and Indoor Air. (2000) "Clearing the Air", Washington, National Academy Press.

Comtois, P. and Garcia, B.E. (1994) "Indoor mycology the Northern American Experience", in: Singh, J (Edited by), *Building Mycology, Management of Health and Decay in buildings*, London, E & FN Spon, pp. 260-280.

Crowther, D., Horwood, J., Baker, N., Thomson, D., Pretlove, S., Ridley, I., Oreszczyn, T., (2000), "House Dust Mites and the Built Environment: a Literature Review", available online at: www.arct.cam.ac.uk/research/mite/review.pdf.

Crowther, D., Pretlove, S., Ridley, I., Oreszczyn, T., Wilkinson, T. (2002) "A Hygrothermal Model of House Dust Mite Response to Environmental Conditions in Dwellings Summary Report", available online at: http://www.arct.cam.ac.uk/research/mite/four.pdf.

Cuijpers, C.E.J., Swaen, G.M.H., Wessling, G., Sturmans, F., Wouters, E.F.M. (1995) "Adverse effects of the indoor environment on respiratory health in primary school children", *Environ Res*, Vol. 68, pp. 11-23.

Cunningham, M. J. (1996) "Controlling Dust Mites Psychrometrically - a Review for Building Scientists and Engineers", *Indoor Air*, Vol. 6, pp. 249-258.

Dales, R., Zwanenburg, H. and Burnett, R. (1990) "The Canadian air quality health survey: Influence of home dampness and moulds on respiratory health", in: *Proceeding of Indoor air '90. 5th international conference on Indoor air quality and climate,* Ottawa, Indoor Air 90, Vol. 1, pp 145-147.

Department of Environment, Transport and Regions, (1996) "English House Condition Survey 1996", London, DETR.

Emenius G., Svartengren M., Korsgaard J., Nordvall L., Pershagen G. and Wickman M. (2004) Building characteristics, indoor air quality and recurrent wheezing in very young children (BAMSE), Idoor Air Vol. 14(1) pp. 34 - 42.

Emenius, G., Egmar, A., Wickan, M. (1998) "Mechanical ventilation protects one-storey single-dwelling houses against increased air humidity, domestic mite allergens and indoor pollutants in a cold climatic region", Clinical *and Experimental Allergy*, Vol. 28(11), pp. 1389-96.

Flannigan, B., MaCabee, E.M., McGarry, F. and Strachan, D.P. (1990) "Wheeze in children: An investigation of air spora in the home", in: *Indoor air '90. 5th international conference on Indoor air quality and climate,* Ottawa, Indoor Air 90, Vol. 2 pp. 27-32.

Fletcher, A.M., Pickering, C.A., Custovic, A., Simpson, J., Kennaugh, J., Woodcock, A. (1996), "Reduction in humidity as a method of controlling mites and mite allergens: the use of mechanical ventilation in British domestic dwellings", *Clinical and Experimental Allergy*, Vol. 26(9), pp.1051-6.

Garrett, M.H., Rayment, P.R., Hooper, M.A., Abramson, M.J., Hooper, B.M. (1998) "Indoor airborne fungal spores, house dampness and associations with environmental factors and respiratory health in children", *Clinical and Experimental Allergy*, Vol. 28, pp. 459-467.

Gotzsche, P.C., Hammrquist, C., Burr, M. (1998) "House dust mite control measures in the management of asthma: meta-analysis", *British Medical Journal*, Vol. 317, pp.1105-1110.

Hagerhed, L., Bornehag, C.G., Sundell J. (2002) "Dampness in buildings and health. Building characteristics as predictors for dampness in 8681 Swedish dwellings", in: *Proceedings: Indoor Air 2002. 9th International Conference*

on Indoor Air Quality and Climate, Monterey, Indoor Air 2002.

Hart, B.J. and Whitehead, L. (1990) "Ecology of house dust mite in Oxfordshire", *Clinical and Experimental Allergy*, Vol. 20, pp. 203-209.

Harving, H., Korsgaard, J., Dahl, R. (1994) "Clinical efficacy of reduction in house-dust mite exposure in specially designed, mechanically ventilated "healthy" homes", *Allergy*, Vol. 49(10), pp.866-70.

Howieson, S.G., Lawson, A., McSharry, C., Morris, G., McKenzie, E. (2002) "1B2O5 Indoor Air Quality, Dust Mite Allergens And Asthma", in: *Proceedings Indoor Air 2002. 9th International Conference on Indoor Air Quality and Climate*, Monterey, Indoor Air 2002, pp.113-118.

Howieson, S.G., Lawson, A., McSharry, C., Morris, G., McKenzie, E., Jackson, J. (2003) "Domestic ventilation rates, indoor humidity and dust mite allergens - Are our homes causing the asthma pandemic", *Building Services Engineering, Research and Technology Journal*, Vol. 24, No. 3, pp.137-147.

Htut, T., Burgess, I.F., Maunder, J.W., Basham, E. (1996) "A pilot study on the effect of one room mechanical ventilation with heat recovery (MVHR) units on house dust mite populations and Der p 1 levels in laboratory simulated bedrooms and on ambient conditions in an occupied bedroom in Cambridge, UK", *International Journal of Environmental Health Research*, Vol. 6, pp.301-313.

Hunter, C.A., Grant C., Flannigan B. and Bravery A.F. (1988) "Mould in buildings: the air spora of domestic buildings", *International Biodeterioration*, Vol. 24, pp. 81-101.

Hunter, C.A., Hull A.V., Higham, D.F., Grimes, C.P. and Lea, R.G. (1996) "Fungi and bacteria" in: Berry, R. W., Brown, V. M., Coward, S. K.D., Crump, D. R., Gavin, M., Grimes, C. P., Higham, D. F., Hull, A. V., Hunter, C. A., Jeffery, I. G., Lea, R. G., Llewellyn, J. W. and Raw, G. J., (1996), *BRE Report 299. Indoor air quality in homes: part 1*, Garston, BRE; pp 97-115.

Husman, T., Koskinen O., Hyvarinen A., Reponen T., Ruuskanen J. and Nevalainen A. (1993) "Respiratory symptoms and infections among residents in dwellings with moisture problem or mould growth", in: *Proceedings of indoor air 93; 6th international conference on indoor air quality and climate*, Vol 1, pp171-174.

Irie, T., Hasegawa, Y. and Yoshikawa, M. (1990) "Measurement of living mites in private wooden dwellings, Japan", in: *Proceedings International Conference on Indoor Air Quality and Climate*, Ottawa, Vol. 2, pp 61-66.

ISBE (International Society of the Built Environment), (2003) "Workgroup Report", *Indoor and Built Environment*, Vol. 12(4), pp. 259-264.

Kalliokoski, P. (2003) "Risks caused by airborne microbes in hospitals – source control is important", *Indoor-Built Environ.*; Vol. 12, pp. 41-46.

Kemp, P.C., Neumeister-Kemp H.G., Kock, C., Lysek, G., Murray, F. (2002a) "Determining the Growth and Vitality of Micro-Organisms in Carpets and Mattresses in Non-Problem Dwellings by Measuring CO2 Released during Respiration", *Indoor-Built Environ*, Vol.11, pp.214-220.

Kemp, P.C., Neumeister-Kemp H.G., Murray, F., Lysek, G. (2002b) "Airborne Fungi in Non-Problem Buildings in a Southern-Hemisphere Mediterranean Climate: Preliminary Study of Natural and Mechanical Ventilation", *Indoor-Built Environ.*, Vol. 11, pp. 44-53.

Korsgaard, J. (1979) "The effect of indoor environment on house dust mite", in: P O Fanger and O Valbjorn, (Edited by), *Indoor Climate*, Copenhagen, Danish Building Research Institute, pp 187-199.

Lacey, J. (1994) "Indoor aerobiology and health", in: Singh, J., (Edited by), *Building Mycology, Management of Health and Decay in buildings*, London, E & FN Spon, pp. 77-130.

Lange, J.H., Grad, J.W., Lange, P.A., Thomulka, K.W., Dunmyre, G., Lee, R.J., Richardson, C.F., and Blumershire, R.V.H. (1993) "Asbestos abatement of ceiling panels and mould growth in a public school building after water damage: a case study", *Fresenius Environ Bulletin*, Vol. 2, pp. 13-18.

Leeuwen, W.S. (1924) "Bronchial asthma in relation to climate", *Proc Roy Soc Med*, pp. 17-19.

Li, C.S. and Hsu, L.Y. (1996) "Home dampness and childhood respiratory symptoms in a subtropical climate", *Arch Environ Health*, Vol. 51, pp. 42-46.

Lowe, R. (2000) "Psychrometric control of Dust mites in UK Housing", *Building Services Engineering Research and Technology*, Vol. 21, No. 4, pp.274-276.

Lugauskas, A., Krikstapois, A., Seskauskas, V. (2003) "Species of conditionally pathogenic Micromycetes in the air of dwelling and occupational premises", *Indoor-Built Environ*, Vol.12, pp. 167-178.

McIntyre, D.A. (1992) "The control of house dust mites by ventilation: a pilot study", *Proc. 13th AIVC Conf.*, Nice, AIVC, pp.497-507.

Menetrez, M.Y., Forde K.K., DeJesus V.R. (2002) "Testing Antimicrobial Efficacy on Porous Materials", *Indoor-Built Environ.*, Vol.11, pp. 202-207.

Menetrez, M.Y., Foarde, K.K. (2002) "Microbial Volatile Organic Compound Emission Rates and Exposure Model", *Indoor-Built Environ.*, Vol. 11, pp. 208-213.

Minnesota Department of Health (2001) "Recommended best practices for mould investigations in Minnesota schools", Environmental Health Division, Indoor Air Unit, St. Paul, Mn 55164.

Mussalo-Rauhamaa, H., Suomalainen, H., Helin, T., Elg, P., Orpana, A. (2003) "Bronchoalveolar lavage findings from persons exposed to moulds in water-damaged houses", *Indoor-Built Environment*, Vol. 12, pp. 237-237.

New York City Department of Health (2000) "Guidelines on assessment and remediation of fungi in indoor environments", New York, Bureau of Environmental and Occupational Disease Epidemiology.

Niven, R., Fletcher, A.M., Pickering, A.C., Custovic, A., Sivour, J.B., Preece, A.R., Oldham, L.A., Francis, H.C. (1999) "Attempting to control mite allergens with mechanical ventilation and dehumidification in British houses", *Journal of Allergy Clinical Immunology*, Vol. 103 (5 Pt 1), pp. 756-62.

Office of the Deputy Prime Minister (ODPM), (2003), "English House Condition Survey 2001", ODPM, London.

Peat, J.K., Dickerson, J., Li, J. (1998) "Effects of damp and mould in the home on respiratory health: a review of the literature", *Allergy*, Vol. 53, pp. 120-128.

Platts-Mills, T.A.E. and De Weck, A.L. (1989) "Dust mite allergens and asthma: A world wide problem", *J Allergy Clin Immunol.*, Vol. 83, pp. 416-27.

Pope, A.M., Patterson, R. and Burge, H. (Edited by) (1993) "Indoor allergens: assessing and controlling adverse health effects", Washington, National Academy Press.

Rao, C.Y., Burge, H.A., Chang, J. (1996) "Review of quantitative standards and guidelines for fungi in indoor air", *J Waste Manag Assoc.*, Vol. 46, pp. 899-908.

Rautuiala, S., Reponen, T., Nevalainen, A., Husman, T., Kalliokoski, P. (1998) "Control of exposure to airborne viable microorganisms during remediation of mouldy buildings: report of three case studies", *Am J Ind Hyg.*, Vol. 59, pp. 455-460.

Raw, G.H. and Fox, T.A. (1990) "Condensation heating and ventilation in small homes", in: *CIB Conference. Energy moisture and climate*, Rotterdam, CIB.

Raw, G.J., Aizlewood, C.E. and Hamilton, M. R. (2001) "Building Regulation, Health and Safety", Garston, BRE.

Ridley, I., Fox, J., Oreszczyn, T., Houng, S.H. (2003) "The impact of replacement windows on air infiltration and indoor air quality in dwellings", *The International Journal of Ventilation*, Vol. 1, No. 3, pp. 209-218.

Rylander, R. (2003) "Humid building – the problem", *Indoor and Built Environment.*, Vol. 12, pp. 211-213.

Sarca, S, Asan, A, Otkun, MT, Ture, M. (2002) "Monitoring Indoor Airborne Fungi and Bacteria in the Different Areas of Trakya University Hospital, Edirne, Turkey", *Indoor-Built Environ.*, Vol. 11, pp. 285-292.

Singh, J. (1994a) "Building Mycology Management of Health and Decay in buildings", London, E & FN Spon.

Singh, J. (1994b) "Allergy Problem in Buildings", Lancaster, Quay Publishing.

No.1, pp. 22-34.

Singh, J. (1999) Sick Building Syndrome, Facilities Management, Health and Safety, November issue, pp. 12-15.

Spengler, J., Neas, L., Nakai, S., Dockery, D., Speizer, F., Ware, J., and Raizenne, M. (1993)

"Respiratory symptoms and housing characteristics", in: *Proceedings of indoor air 93; 6th international conference on indoor air quality and climate*, Vol 1, pp. 165-170.

Stephen, F. R., McIntyre, D. A., Lane, A., Raw, G.J., Wiech, C.R., Frederick, J. (1997) "Ventilation and House Air Tightness: Effect on Indoor Temperature and Humidity in Southampton, UK", *BSERT* (Building Services Engineering Research Technology Journal), Vol.18, No.3, pp.141-147.

Strachan, D.P (1998) "House dust mite allergen avoidance in asthma. Benefits unproved but not yet excluded", *British Medical Journal*, Vol. 317, pp.1096-1097.

Su, H.L., Spengler, J.D., and Burge, H.A. (1990) "Examination of microbiological concentration and examination with childhood respiratory health", in: *Proceedings of indoor air 90; 5th Indoor international conference on indoor air and climate,* Ottawa, Indoor Air 90, Vol. 2, pp. 21-26.

Sundell, J., Wickman, M., Pershagen, G., Nordvall, S.L. (1995) "Ventilation in homes infested by house-dust mites", *Allergy*, Vol. 50, No. 2, pp.106-12.

Toma, T., Miyagi, I., Kishimoto, M., Nagama, T., and Tamanaha, I. (1993) "Fauna and seasonal appearances of mites in house dusts collected from the residences including asthmatic children in Okinawa Prefecture, Ryukyu Archipelago", *Japanese Journal of Sanitary Zoology*, Vol. 44, pp. 223-225.

Wargocki, P., Sundell, J., Bischof, W. (2002) "Ventilation and health in non-industrial indoor environments: report from a European Multidisciplinary Scientific Consensus", *Indoor Air*; Vol. 12, No. 2, pp. 113-8.

Warner, J., Ferderick, J., Bryant, T., Weich, C., Raw, G., Hunter, C., Stephen, F., McIntyre, D. and Warner, J. (2000) Mechanical ventilation and high efficiency vacuum cleaning: a combined strategy of mite and mite allergen reduction in the control of mite-sensitive asthma, J.Allergy Clin. Immunol., 106(No. 1, Pt 1), 75-82.

White, J.H. (1990) "Solving moisture and mould problems", in: *Proceedings of indoor air 90; 5th international conference on indoor air quality and climate,* Ottawa, Indoor Air 90, Vol. 4, pp. 589-594.

Williamson, I.J., Martin, C.J., McGill, G., Monie, R.D.H., Fennerty, A.G. (1997) "Damp housing and asthma: a case control study", *Thorax*, Vol. 52, pp. 229-234.

Woolliscroft, M., (1997), "PH-97-8-3. Residential Ventilation in the United Kingdom: An Overview", *ASHRAE Transactions: Symposia,* Vol. 103, No. 1, Paper Number: PH-97-8-3, pp. 706-716.

Zock, J.P., Jarvis, D., Luczynska, C., Sunyer, J., Burney, P. (2002) "Housing characteristics, reported mould exposure, and asthma in the EC respiratory health survey", *Journal of Allergy and Clinical Immunology*, Vol. 110(2) pp. 285-292.

Zureik, M., Neukirch, C., Leynaeret, B., Liard, R., Bousquet, J., Neukirch, F. (2002) "Sensitisation to airborne moulds and severity of asthma: cross sectional study from EC respiratory health survey", *BMJ*, Vol. 325 (7361) pp. 411-414.

Appendix A.0: Published Papers

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A.0.2: *Predicting the population dynamics of the house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle*

Phillip Biddulph[1,5], David Crowther[1], Brian Leung[2], Toby Wilkinson[1,3], Barbara Hart[4], Tadj Oreszczyn[5], Stephen Pretlove[6], Ian Ridley[5], **Marcella Ucci**[5]

[1]The Martin Centre, University of Cambridge, 1 Scroope Terrace, Cambridge CB2 1PX, UK
[2]Biology Department, McGill University, 1205 Docteur Penfield, Montreal, Quebec H3A 1B1, Canada
[3]Medical Entomology Centre, Barrington Road, Shepreth, Royston SG8 6QZ, UK
[4]Royal Agricultural College, Stroud Road, Cirencester, Gloucestershire, GL7 6JS,UK
[5]The Bartlett School of Graduate Studies, University College London, WC1E 6BT, UK
[6]Kingston University, School of Architecture & Landscape, London, KT1 2QJ, UK

Abstract

A generalised model of the life cycle of a house dust mite, which can be tailored to any particular species of domestic mite, is presented. The model takes into account the effects of hygrothermal conditions on each life cycle phase. It is used in a computer simulation program, called POPMITE, which, by incorporating a population age structure, is able to predict population dynamics.

The POPMITE simulation is adapted to the *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) (DP) mite using published data on the egg development period, total development period, adult longevity, mortality during egg development, mortality during juvenile development, and fecundity of individual DP mites held at a range of constant hygrothermal conditions.

An example is given which illustrates how the model functions under constant hygrothermal conditions.

A preliminary validation of POPMITE is made by a comparison of the POPMITE predictions with published measurements of population growth of DP mites held at a range constant hygrothermal conditions for 21 days.

The POPMITE simulation is used to provide predictions of population growth or decline for a wide range of constant relative humidity and temperature combinations for 30 and 60 days.

The adaptation of the model to correctly take account of fluctuating hygrothermal conditions is discussed.

Keywords

House dust mites, population model, relative humidity, temperature, life cycle, population structure

Introduction

The house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) (DP) is one of several species giving rise to allergens that play a major role in allergic disease, especially asthma (Voorhorst et al. 1969 and Ford et al. 1985). DP is the most common species of house dust mite in the UK, and is predominant in many countries around the world (Bronswijk 1981 and Colloff 1998). Their major habitats are beds, carpets and soft furnishings.

Mites have few natural predators, so that in a typical habitat the principal limits on the population size are the available food and the hygrothermal environment, which can be described by the relative humidity (RH) and the temperature (T) (Bronswijk 1981). In this paper we make the simplifying assumption that food is plentiful and is not limiting the population size.

Predicting the population dynamics in the hygrothermal conditions of the habitat is a crucial step in the study of different strategies for controlling mite populations, and therefore the production of allergen (see Crowther et al. 2006). Past, present and simulated future climate data can be used in conjunction with building simulations (for example see EnergyPlus 2004) to predict the internal climate of bedrooms for a wide range of house types and conditions, as well as different occupant behaviour regimes. These predictions or actual measurements can be used in turn to predict the internal conditions of beds (Pretlove et al. 2005).

An accurate computer simulation of the population dynamics of dust mites is needed to complement the range of simulations available to predict the environment of the habitat. The suite of simulations can then be used to test scenarios for controlling dust mite populations in beds. Small changes in occupant behaviour or building design may have a significant effect on the population dynamics.

Previous attempts to model the population dynamics by Cunningham (2000) and more recently Crowther et al. (2006) have used the simplifying assumption that there exists a population growth multiplication factor which is constant for a particular RH and T combination, regardless of the structure of the population. Whilst these models have the advantage of being easy to implement they can lead to misleading results even during periods of constant conditions. For example a population of pure eggs is unable to grow until at least one mating pair has matured and as a population develops the numbers of egg laying females will fluctuate naturally. This would lead to fluctuating rates of population growth, which would cause problems for these models, both in terms of calibration and use. To overcome them a model which can keep track of the population structure has been developed.

Mites develop through various life phases, from eggs to juveniles (larvae, protonymphs and tritonymphs for DP) to adults. Mites at each phase of the life cycle have different development times and mortalities depending on the hygrothermal

conditions (Gamal Eddin et al. 1983a,c). The fecundity of female adults also depends on the hygrothermal conditions (Gamal Eddin et al. 1983b).

The population dynamics of dust mites is most sensitive to the parameters described above. As with any model, this model uses simplifying assumptions, particularly for the less sensitive parameters (e.g. the age dependence of the egg laying rate). A preliminary validation of the model indicates that these assumptions are acceptable.

A generalised model of the life cycle of a house dust mite, using information on development times and mortalities, can be tailored to any particular species of domestic mite. The age structure of the mite population can be simulated by keeping track of the development and numbers of mites in batches as they progress through the life cycle model.

In this way a computer simulation program able to predict population dynamics, called POPMITE, has been constructed that incorporates the life cycle model and a population age structure. POPMITE has been tailored to DP mites using a selection of published data describing their physiological response to a range of constant hygrothermal conditions.

Nomenclature

The following table lists the symbols, their meanings and units as used throughout this paper.

Symbol	Meaning	Units
T	Temperature	°C
RH	Relative humidity	%
t	Time slice	hour
N_{egg}	Number of eggs	eggs
$N_{juvenile}$	Number of juveniles	juveniles
N_{adult}	Number of adults	adults
D_{egg}	Egg development duration	ours
$D_{juvenile}$	Juvenile development duration	hours
D_{adult}	Adult longevity	hours
R_{egg}	Egg Percentage Development rate	%/hour
$R_{juvenile}$	Juvenile Development rate	%/hour
R_{adult}	Adult Ageing rate	%/hour
d_{egg}	Percentage development of eggs	%
$d_{juvenile}$	Percentage development of juveniles	%
d_{adult}	Percentage development of adults	%
M_{egg}	Total mortality during the egg phase.	%
$M_{juvenile}$	Total mortality during the juvenile phases.	%
M_{adult}	Total mortality during the adult phase.	%
s_{egg}	Egg survivability, the probability of surviving until hatching.	
$s_{juvenile}$	Juvenile survivability, the probability of a freshly hatched juvenile surviving until moulting into an adult.	
s_{adult}	Adult survivability, the probability of surviving the natural adult life span.	
S_{egg}	Egg hourly survival probability	$hour^{-1}$
$S_{juvenile}$	Juvenile hourly survival probability	$hour^{-1}$
S_{adult}	Adult hourly survival probability	$hour^{-1}$
F	Egg laying rate of adult female mites.	eggs/hour
f	Fecundity or the mean number of eggs produced per female mite during her adult life.	eggs

Theory

The methods for calculating the population dynamics described here are similar to those that use the Leslie matrix model (Leslie 1945) as described by Nisbet and Gurney (1982) and Case (2000).

If the initial structure of a population of mites (numbers and ages of eggs, juveniles and adults) is known, then our aim is to predict the change in the structure after a short period of constant hygrothermal conditions. This can be done by calculating:

- the increase in development and the loss of mites for each of the life cycle phases separately, and
- the number of freshly laid eggs.

By repeating this process over many time periods one can thus predict the population dynamics.

A population of mites will consist of batches of individuals at any one of the phases of their life cycle. Each batch will be at a unique development stage within the phase and will be progressing through the phase at a rate which depends on the hygrothermal environment. The environment will also play a role in determining the chances of survival and the rate at which adult females lay eggs. The physiological response of mites in a particular phase to the constant conditions can be described by:

- the percentage development rate (R),
- the survival probability (S), and
- for adult females, the egg laying rate (F).

It is assumed that each of these parameters R, S and F will depend only on the current hygrothermal conditions RH and T.

In the model we can make a list, or an array, of the number of mites in each batch in each phase, indexed by the percentage development $d\{t_n\}$, where t_n is the current time. So the number of mites in a batch in a phase is described as $N\{d\{t_n\}\}$, where $d\{t_n\} = 0\%$ at the beginning of the phase. To calculate how further developed this batch of mites are in the next time slice t_{n+1}, where $t_{n+1} = t_n + t_{unit}$ and t_{unit} is typically 1 hour, we use the percentage development per hour $R(RH,T)$. So the new percentage development of this batch of mites at time t_{n+1} is:

$$d\{t_{n+1}\} = d\{t_n\} + R(RH,T) \qquad (1)$$

During this time some of the mites in the batch will die. To calculate how many mites survive into the next time slice we use the survival probability per hour $S(RH,T)$. So the number of mites surviving into the next time slice is given by:

$$N\{d\{t_{n+1}\}\} = N\{d\{t_n\}\} \times S(RH,T) \qquad (2)$$

It should be noted that $N\{d\{t_{n+1}\}\}$ is not an integer and can become very much less than 1 if the conditions are bad for an extended period of time. This can be interpreted as

the probability of survival from eradication during this time slice. If $N\{d\{t_{n+1}\}\}$ is tiny (less than say 0.000001) then it is very unlikely that this batch will survive and it is removed from the list of batches.

The percentage development rate and survival rate are used in every phase of the life cycle and a subscript is now used to indicate which phase a particular parameter represents. For example $N_{egg}\{d_{egg}\{t_n\}\}$ is the number of eggs with percentage development $d_{egg}\{t_n\}$, at time t_n.

The adult phase is a special case, as in addition to the development rate and the survival rate, adult female mites may lay eggs. To calculate how many eggs are laid in the next time slice we use the eggs laid per hour $F(RH,T)$. So the number of fresh eggs laid in the next time slice is given by:

$$N_{egg}\{d_{egg}\{t_{n+1}\}\} = N_{female} \times F(RH,T), \qquad (3)$$

where $d_{egg}\{t_{n+1}\} = 0\%$, because they are freshly laid, and $N_{female} = N_{adult}/2$

It is assumed for the purposes of this model that half the adults are female (Hodgson (1976)) and that all adult females of all ages can produce eggs if the conditions are favourable. The sex ratio of eggs produced is assumed to be 1:1, and there are no differences between the sexes, other than that female mites can lay eggs. These simplifying assumptions can be relaxed if necessary.

This process is repeated each time slice with adult female mites replenishing the supply of eggs, as batches of mites progress through the phases, getting ever more developed, as well as ever more depleted in numbers as they die, until they reach a development of 100%. Once a batch of mites has reached a development of 100%, it is moved into the next phase with a percentage development of 0%. At the end of the final adult phase the mites die and are removed from the model completely.

Figure 1 is a schematic diagram showing the model of a simplified mite life cycle with only three phases, egg, juvenile and adult. Time proceeds down the page for three consecutive time slices. The RH and T are assumed to be constant throughout each time slice. The structure of each phase is depicted as separate histograms across the page with development as the x-axis and number of mites as the y-axis. Within the histogram, batches are represented by thick vertical bars. The height of each bar represents the number of eggs, juveniles or adults in a batch, and the position along the histogram shows its progress. After each time slice, batches are moved along the histograms until they reach the end and are then either moved into the next stage or, for adults, are removed altogether, representing the end of their lifespan. The bars shrink in size at every time slice depending on the survival rates. The female adults lay fresh eggs which form a new batch at the start of the next egg histogram as indicated by the dotted line.

The model naturally introduces a mix or structure into the population. The population of mites at any one moment will consist of eggs, juveniles and adults, all at different stages of development and age. The exact structure will depend on the history of the population and, to get a realistic prediction of the population dynamics over an extended period of time, it is important to keep track of this structure. For example a population experiencing a period of bad conditions leading to a much depleted population will respond very differently to a period of good conditions than a healthy population would.

The POPMITE simulation

To use the model in a simulation of a population of a real mite species, it is necessary to have values for the percentage development rate (R), the survival rate (S) for each phase in a mite's life cycle and the egg laying rate for adult females (F) for a wide range of RH and T combinations.

Data from a number of different sources has already been published describing the physiological response of the different phases of individual DP mites to a range of constant RH and T combinations. A selection of this data, suitably analysed, has been used to create a rough simulation. The data comes from different experimental groups, which use different experimental methodologies that may not be compatible. It is therefore difficult to extract anything more than an estimate of the physiological response to the hygrothermal conditions. Indeed in analysing the data our primary intention at this time is not to derive accurate relationships, but to provide data to illustrate the model. We therefore have not provided measures of the quality of the fits to the data. The authors of this paper are collecting a fuller and more consistent set of data to be used by the simulation which will potentially give a more precise prediction. The data and a description of an improved simulation will be published at a later date.

From the published data for the model we require the mathematical descriptions or formulae of the parameters:

- $R_{egg}(RH,T)$, $R_{juvenile}(RH,T)$, $R_{adult}(RH,T)$,
- $S_{egg}(RH,T)$, $S_{juvenile}(RH,T)$, $S_{adult}(RH,T)$, and
- $F(RH,T)$.

Egg development rate $R_{egg}(RH,T)$

No direct measurement of the development rate of eggs $R_{egg}(RH,T)$ was found in the literature. There is however quite detailed information on the development duration of eggs for a wide range of RH and T combinations from which the development rate can be calculated. Table 1 shows the development duration in days of DP eggs held at constant RH and T conditions.

For the purposes of the model we make the simplifying assumption that for any given combination of RH and T the development rate is constant throughout the egg life stage. We can then extract the percentage development per hour as $R_{egg} = 100\%/D_{egg}$,

where D_{egg} is the development duration in hours calculated from the data recorded in days in Table 1. Figure 2 shows R_{egg} plotted against temperature.

A linear function of temperature can be used to describe the egg development rate. Below about 50% RH no data was found on the development duration and above 50% RH no obvious correlation was found with RH (i.e. the development rate did not vary significantly for different RH values above 50% for fixed values of temperature).

The solid line on Fig. 2 is a fit to the egg development data and gives a rate of 0.91 % development per day per °C. In other words, for every 1°C increase in temperature the development rate increases by 0.91 of a percent per day. If we assume that we can extrapolate this function to lower temperatures, then development should stop completely below 9°C.

Equation 4 is the fit to the data and gives the development rate formula for $R_{egg}(RH,T)$ in % development per hour, to be used in the model.

$$\begin{aligned} R_{egg}(RH,T) &= 0.91\times(T-9)/24 \quad T>9°C \\ R_{egg}(RH,T) &= 0 \qquad\qquad\quad T<9°C \end{aligned} \tag{4}$$

The formula for the development duration of eggs will be useful later for the calculation of the juvenile development rate and the egg survival rate. This is given by equation 5, where $D_{egg}(RH,T)$ is in hours,

$$\begin{aligned} D_{egg}(RH,T) &= \frac{24\times100}{0.91\times(T-9)} \quad T>9°C \\ D_{egg}(RH,T) &= \infty \qquad\qquad\quad T<9°C \end{aligned} \tag{5}$$

Juvenile development rate $R_{juvenile}(RH,T)$

No direct measurement of the development rate of the juvenile phases of the DP mite was found in the literature. However some information for the total development duration from fresh eggs to adults is available and, by subtracting the egg development duration, the development duration of juveniles can therefore be inferred. Table 2 shows the total development duration for different combinations of RH and T. The data is from many sources, and is concentrated along the 75%RH row and the 25°C column. It can be seen that the lower the temperature, the longer the mites take to develop.

As an intermediate step we need to establish a development rate for the combined egg and juvenile life stages. In order to do so we make the simplifying assumption that, for any given combination of RH and T, the development rate is constant throughout the combined egg and juvenile development life stage. Then we can extract an percentage development per hour as $R_{total}=100\%/D_{total}$, where D_{total} is the

development time in hours calculated from the data recorded in days in Table 2. Figure 3 shows R_{total} plotted against T.

As with eggs, a linear function of the temperature can be used to describe the combined egg and juvenile development rate. Below about 50% RH no data was found on the development duration and above 50% RH no obvious correlation with development rate was found with RH. This may seem surprising given that water balance is important to mites in that above a threshold RH level they extract moisture from the atmosphere and without this moisture the mites eventually die (Arlian 1992). Mites can survive for a short period of time when the RH is below this threshold and can then recover once the RH goes above the threshold (de Boer et al. 1998). In conditions which oscillate above and below the threshold the mite development duration will depend on the length of exposure and absolute level of the low RH conditions. However, in constant conditions with RH below the threshold, mites never get the opportunity to recover and therefore die.

The solid line is a fit to the data and gives a rate of 0.33 % total development per day per °C. For every 1°C increase in temperature the development rate increases by 0.33 of a percent per day. If we assume that we can extrapolate this function to lower temperatures, then the development should stop completely below about 13°C.

$$\begin{aligned} R_{total}(RH,T) &= 0.33\times(T-13)/24 \quad T>13°C \\ R_{total}(RH,T) &= 0 \qquad\qquad\quad T<13°C \end{aligned} \tag{6}$$

The formula for overall development time from eggs to adults is given by,

$$\begin{aligned} D_{total}(RH,T) &= \frac{24\times100}{0.33\times(T-13)} \quad T>13°C \\ D_{total}(RH,T) &= \infty \qquad\qquad\quad T<13°C \end{aligned} \tag{7}$$

We can now subtract the egg development duration from the total development duration to get the juvenile development duration.

$$D_{juvenile}(RH,T) = D_{total}(RH,T) - D_{egg}(RH,T) \tag{8}$$

The juvenile development rate is therefore

$$R_{juvenile}(RH,T) = 100\%/D_{juvenile}(RH,T) \tag{9}$$

Adult ageing rate $R_{adult}(RH,T)$

The adult ageing rate is the rate at which both male and female adults progress towards the end of their natural life span. No direct measurements of this have been found. However table 3 gives the longevity in days of DP adults held at constant RH and T conditions.

The very sparse data there is, shows that adult mites have a much shorter life at low RH than at high RH. Figure 4 shows adult longevity as a function of temperature with two fits to the data.

The longevity of adult mites can therefore be modelled with the following equations with an arbitrary split at 60% RH. More data is needed to give a better representation of the real response of adult longevity to RH and T.

$$\begin{aligned} D_{adult}(RH,T) &= 24\times(61.42\text{-}T\times 1.31) \quad RH > 60\% \\ D_{adult}(RH,T) &= 24\times(16.44\text{-}T\times 0.32) \quad RH < 60\% \end{aligned} \tag{10}$$

The longevity data can be used to calculate the rate of ageing in % ageing per hour.

$$R_{adult}(RH,T) = 100\% / D_{adult}(RH,T) \tag{11}$$

Egg survival rate during development $S_{egg}(RH,T)$

No data for the survival rates of mite eggs during their development phase was found. However there is data on the mortality (M_{egg}) at the end of the development phase. This is defined as the percentage of mite eggs which die during the period of development and is given by this formula,

$$M_{egg} = \frac{N_{egg} - N_{juvenile}}{N_{egg}} \times 100\% \tag{12}$$

Table 4 shows the egg mortality, in percent, during the development period of mite eggs to juveniles.

The mortality is highest at low temperatures whatever the RH and also at low RH at high and low temperatures. There is clearly some discrepancy between the different datasets, especially at 25°C and 75% and 80% RH. For RH below an arbitrary cut-off, which by coincidence is also 60%, a simple quadratic of the temperature is used,

$$M_{egg}(RH,T) = 195.78 - 14.35T + 0.32T^2 \quad RH < 60\% \tag{13}$$

At RHs above 60% a more complicated exponential of the temperature is used with a simple linear formula for the RH,

$$M_{egg}(RH,T) = 31.20 - 0.19RH + 2754.05e^{-0.37T} \quad RH > 60\% \tag{14}$$

Figure 5, Figure 6 and Figure 7 show the fits to the data.

The mortality is equal to a hundred percent minus the survivability,

$$M_{egg}(RH,T) = 100\% - s_{egg} \tag{15}$$

The survivability is the product of all the hourly survival rates during the development duration, and is therefore given by

$$s_{egg} = S_{egg}{}^{D_{egg}(RH,T)/t_{unit}} \tag{16}$$

This leads to an hourly survival rate, which can be used in the model.

$$S_{egg}(RH,T) = \left(1 - \frac{M_{egg}(RH,T)}{100\%}\right)^{t_{unit}/D_{egg}(RH,T)} \tag{17}$$

It is important to note that the hourly survival rate depends on the development duration as well as the mortality of eggs.

Survival rate during the juvenile phases $S_{juvenile}(RH,T)$

No data for the survival rates of juvenile mites during their development phase was found. To calculate the juvenile hourly survival rate, the total mortality during the overall mite development from egg to adult is used. It is defined, in a similar way to the mortality during the egg development period, as the percentage of mites which die during the overall period of development and is given by this formula:

$$M_{total} = \frac{N_{egg} - N_{adult}}{N_{egg}} \times 100\% \tag{18}$$

Table 5 shows the total mortality, in percent, during the development of mites from fresh eggs to adults.

The data is concentrated along the 75% RH row and the 25°C column. The mortality increases at low and high RH, and also at low and high temperatures. The minimum mortality is at 80% RH and 25°C. Two independent quadratic curves are used to fit the RH and the temperature data separately and are then combined together assuming there are no cross correlations.

$$M_{total}(RH,T) = 803.59 - 15.97RH + 0.11RH^2 + 14.14T + 0.27T^2 \tag{19}$$

Figures 8 and 9 shows the data and the fits.

The probability of mortality during the complete development period is equal to the product of the probability of mortality during the egg development phase and the juvenile phase.

$$\frac{M_{total}(RH,T)}{100\%} = \frac{M_{egg}(RH,T)}{100\%} \times \frac{M_{juvenile}(RH,T)}{100\%} \tag{20}$$

Using the same logic from the previous section on the survival rate during the egg development phase, the hourly survival rate of juveniles is given by:

$$S_{juvenile}(RH,T) = \left(1 - \frac{M_{juvenile}(RH,T)}{100\%}\right)^{t_{unit}/D_{juvenile}(RH,T)} \tag{21}$$

Substituting equation 20 into equation 21 gives

$$S_{juvenile}(RH,T) = \left(1 - \frac{M_{total}(RH,T)}{M_{egg}(RH,T)}\right)^{t_{unit}/D_{juvenile}(RH,T)} \tag{22}$$

Survival rate during the adult phase $S_{adult}(RH,T)$

Since no direct or indirect measurements of the survival rates or mortality for adults have been published, we have assumed that the mortality of adults is the same as the mortality of juveniles.

$$M_{adult} = M_{juvenile} = \frac{M_{total}}{M_{egg}} \times 100\% \tag{23}$$

This leads to the rate of adult mites surviving per hour as:

$$S_{adult}(RH,T) = \left(1 - \frac{M_{adult}(RH,T)}{100\%}\right)^{t_{unit}/D_{adult}(RH,T)} \tag{24}$$

Substituting equation 23 into equation 24 gives:

$$S_{adult}(RH,T) = \left(1 - \frac{M_{total}(RH,T)}{M_{egg}(RH,T)}\right)^{t_{unit}/D_{adult}(RH,T)} \tag{25}$$

Adult female egg laying rate $F_{adult}(RH,T)$

The fecundity or total mean number of eggs produced by a female mite during her adult life is given in Table 6 in number of eggs.

A cubic formula for both RH and temperature is used to describe the data,

$$f = 336.71 - 65.08RH + 1.16RH^2 - 0.0064RH^3 + 89.02T - 3.04T^2 + 0.032T^3 \tag{26}$$

Figures 10 and 11 show the data and the fits.

The simplifying assumption has been made that female adults lay eggs at a constant rate throughout their adult lifetime. Therefore the probability that they will lay eggs in any one time slice is given by:

$$F(RH,T) = f \times t_{unit} / D_{adult}(RH,T) \quad (27)$$

Using the POPMITE Simulation

The POPMITE simulation program has been written in PERL (www.perl.com). POPMITE can read the hygrothermal conditions, together with a description of the initial structure of the mite population, and predict the population dynamics.

To illustrate how the model functions, POPMITE has been used to predict the population dynamics of 100 freshly laid eggs (no juveniles or adults at the start) in an arbitrarily chosen constant climate of 75%RH and 35°C for 30 days. Figure 12 shows the results.

The plot shows the number of mites as a function of time. Initially there are 100 eggs at the beginning of the first day. Over the next 4 days (the development period at 35°C) the number of eggs has decreased to roughly 80 % (the survival probability). At this point all the eggs turn into juveniles and the number of eggs drops to zero. After approximately 10 further days the number of juveniles has decreased to 60% approximately to leave about 45 juveniles. At this point the juveniles turn into adults and the number of juveniles drops to zero. Immediately the adults start to lay new eggs. Fresh batches of new eggs are then laid every time slice, depending on the number of adults. The number of adults gradually decreases, according to the survivability rate. However after 4 further days the first of the new eggs to be laid start to hatch into juveniles, and the number of juveniles starts to increase. The number of eggs in the population is then equal to the number of eggs being laid minus the number of eggs dying, minus the number of eggs changing into juveniles, and so the egg population starts to decrease. After a further 10 days the juveniles start to turn into adults, and there is consequently a sharp drop in the number of juveniles, a sharp rise in the number of adults and also a sharp rise in the number of eggs. At 29 days the number of adults drops sharply before the upward trend resumes. These are the adults from the original batch of eggs dying.

If this simulation is left to run for any length of time the population of mites will quickly become enormous, which is to be expected for these relatively favourable conditions, with unlimited food supplies.

Preliminary validation by comparison of POPMITE predictions with measurements of mite population growth.

The DP mite population growth and decline, after a 21 day period, under a range of constant RH and temperature conditions, have been measured as reported by

Crowther et al. (2006). A population growth multiplication factor was calculated for each RH and temperature combination by comparing the final population of juveniles and adults with the initial population of juveniles and adults. The population of eggs was not measured but as the initial populations were mature and well mixed it was assumed that there were a similar number of eggs in each sample. No measurement, other than the combined total number of juveniles and adults, was made of the initial population structure.

POPMITE can be used to predict the measurements of the population growth factor. With no other information on the starting population structure, we assume a population structure of 1:1:1 of eggs, juveniles and adults with a spread over all ages. The predictions and data with statistical error bars from Crowther et al (2006) are shown as a function of RH in figures 13, 14, 15, 16 and 17 for fixed temperatures of 15°C, 20°C, 25°C, 30°C and 35°C respectively. The discontinuity at 60% RH in the POPMITE predictions in figures 13 to 17 are due to the use of the arbitrary step change at 60%, as explained earlier, in equation 10 to describe adult longevity and equations 13 and 14 to describe egg mortality.

The POPMITE model under-predicts the growth factor at high RH when the temperature is 30°C, but otherwise the predictions and measurements show a good agreement with each other over the whole RH and T range.

A similar experiment as described by Crowther et al with more detailed measurements of the initial and final population structures should be able to provide a more comprehensive validation of POPMITE under constant conditions. However the very good agreement with the present data gives confidence in POPMITE and allows for a preliminary validation.

Predicting mite population growth at a range of constant conditions

The POPMITE simulation program can now be used to predict mite population growth or decline for a wide range of constant RH and T combinations. Figure 18 shows the predicted growth of a population consisting of 100 freshly laid eggs after 30 days. The thick contour represents the RH and T combinations where the complete population (eggs, juveniles and adults) is numerically equal to the starting population. Figure 18 show a central 'island' of growth with a maximum growth factor of 11.476 (538.8 Eggs, 553.9 Juveniles and 54.9 Adults) at 29°C and 76%RH. Outside this island the populations are in decline, with a higher decline at higher temperatures.

The discontinuity at 60% RH in Figure 18 is again due to the use of the arbitrary step change at 60% in equation 10 to describe adult longevity and equations 13 and 14 to describe egg mortality.

Figure 18 shows the population growth for only 30 days, which means that for populations at low temperatures the mites have not yet completed a full life cycle. At temperatures below 13°C eggs will not have had time to hatch into juveniles, and below about 24°C juveniles will not have had time to turn into adults. The lower

bound of the 'island' of growth at 24°C as shown in Figure 18 is due to mites not reaching maturity and therefore being unable to produce eggs to increase the population.

Figure 19 shows the predicted growth of a population consisting of 100 freshly laid eggs after 60 days. The maximum growth factor of 365.293 (20,002.9 Eggs, 13,062.7 Juveniles and 3,463.7 Adults) is now at 31°C and 76%RH, which is at a slightly higher temperature than the predicted maximum of 29°C for the 30 day simulations. It is also massively increased. Mites held in these conditions are unlikely to reach such numbers unless there is an unlimited amount of food and space available to them. Wilkinson et al. (2002) have shown that 1 gram of food is enough for a population of about 12,000 mites (juveniles and adults) to develop after 18 weeks from only 2 mating pairs held at 25°C and 75%RH. The population then gradually declined as the food was consumed. A limit on the population growth based on the available food can be incorporated into POPMITE if needed. This will be the subject of a future paper by the authors.

The area over which the population is either stable or growing is now much larger. However the lower bound of this growth 'island' is again due to the fact that below 18°C juveniles will not have been able to turn into adults.

The total mortality as described by equation 19 and shown in Figure 8 has a minimum at 26°C and the fecundity as described by equation 26 and Figure 11 has a maximum at 23°C. This would suggest that the maximum growth factor should be found at much lower temperatures than the 29°C for 30 days and 31°C for 60 days. The reason for this apparent discrepancy is that even though the conditions are less favourable, the development rate is very much increased at higher temperatures. The mites are therefore able to replenish their numbers at a faster rate than mites held at lower temperatures.

It is interesting to note that the models based on the constant growth multiplication factor (Crowther et al. 2006) would have predicted a different growth factor after 60 days given the growth after 30 days. Assuming that the growth was constant over the first 30 days then the growth multiplication factor per day would have been $11.476^{(1/30)} = 1.085$. In other words if there where 100 mites then at the end of first day there will be 108.5 mites and at the end second day will be 117.2 mites and so on. This model then predicts a population growth of only $1.085^{60} = 131.699$ after 60 days compared to the POPMITE prediction of 365.293. This illustrates the different results that can be obtained when population structure is taken into account.

Simulation using changing hygrothermal conditions

Once it has been further validated, the model can be used to predict the population dynamics of mites held in constant hygrothermal conditions. Unfortunately the hygrothermal conditions of the typical habitats of mites, such as a mattress, can vary

dramatically. Ridley et al. (2006) have recorded the micro-climatic conditions found throughout the beds of a number of people over a period of a few days. These measurements show that when the bed is empty the mattress is in climatic equilibrium with the bedroom. However once the bed is occupied there is a dramatic increase in the absolute humidity and temperature in the mattress immediately under the body. The temperature increase is usually so high that the corresponding RH drops. Even though the absolute humidity in the bed has increased due to the presence of a sweating, moisture breathing sleeping person, the excess vapour is not enough to increase the relative humidity, since the temperature has risen by a disproportionately large amount at the same time, thus resulting in a drop in relative humidity. For a discussion on this point see Pretlove et al. (2005)

POPMITE can accept continually changing conditions as input, although the quality of the prediction is likely to be degraded the greater the transient nature of the hygrothermal conditions. The reason for this is that at present the model assumes that the survival rates for each phase are constant; in other words the probability of surviving in one time slice does not depend on how long that batch of mites has been exposed to the current environment.

De Boer et al. (1997, 1998) and Pike et al. (2005) have shown that DP populations held in unfavourable conditions of low RH, but given brief periods of favourable high RH conditions every day, are able to survive and even grow. Mite populations held continuously in harsh conditions decline and eventually die out. This suggests that individual mites have a survival probability which depends on how long they are held in harsh conditions.

Arlian and Wharton (1974), Arlian (1975, 1992), Wharton (1978), Arlian and Veselica (1979, 1981a,b) and de Boer (2000) have investigated the water balance of mites (DP and *Dermatophagoides farinae*) and have developed equations which describe the water content of a mite under different hygrothermal conditions. In low RH conditions mites initially lose moisture very quickly, but after a short period of time are able to reduce the water loss drastically, enabling them to survive for an extended period of time. In high RH conditions, mites are able to recover any lost moisture very quickly and to maintain it at high levels to allow them to function and reproduce.

An improvement to the POPMITE model is therefore being developed which will include parameters to track the water contents of mites and therefore reproduce the correct response to conditions fluctuating between high and low RH. Until this is available a minimum survival rate is applied to all batches to allow at least some mites to survive unfavourable conditions. However the resultant predictions are unlikely to be accurate in very transient conditions.

Conclusions

A generalised model of the life cycle of a house dust mite is presented. Previously published data on the physiological effects of exposure of the DP mite to constant

relative humidity and temperature combinations is used to construct a model and computer simulation called POPMITE. For a given starting population structure and climate history, POPMITE is able to predict the most likely population dynamics for the house dust mite DP.

Preliminary validation by comparisons of the POPMITE predictions with measurements of population growth at constant hygrothermal conditions shows good agreement.

Contour plots showing the predicted growth of a population consisting of 100 freshly laid eggs after 30 days and 60 days in a range of RH and T combinations show that temperature plays an important role in controlling the growth and decline of populations.

POPMITE produces very different predictions from models based on the assumption of a constant growth multiplication factor, which are the basis of the models of Cunningham and Crowther. These models are simple and easy to use and can gave a broad indication of the population growth, but they will never be able to give as accurate a prediction as POPMITE, since the population structure and therefore the population growth multiplication factor changes all the time, even under constant conditions.

The current version of POPMITE, when fully validated, will give good predictions of house dust mite population dynamics where conditions are constant or changing slowly, but will tend to give degraded predictions once conditions start to fluctuate. This is still a significant improvement on the models of Cunningham and Crowther, and POPMITE can already be used as part of a study to test scenarios for changing building design or occupant behaviour to reduce mite populations in beds.

A study to provide a precise and comprehensive dataset of the physiological response of DP to a wide range of both constant and varying environmental conditions and diets is under way. This dataset is required to calibrate the parameters needed to track the moisture content of mites for a more accurate version of the POPMITE simulation that will be able to cope fully with varying hygrothermal conditions.

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References

Andersen, A (1988) Population growth and developmental stages of the house dust mite, *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). J. Med Entomol. 25: 370-373

Arlian LG (1975) Dehydration and survival of the European house dust mite, *Dermatophagoides pteronyssinus*. Journal of Medical Entomology, 12/4: 437-442

Arlian LG (1992) Water balance and humidity requirements of house dust mites. Experimental & Applied Acarology, 16: 15-35

Arlian LG, Rapp CM and Ahmed SG (1990) Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Journal of Medical Entomology, 27/6: 1035-1040

Arlian LG and Veselica MM (1979) Water balance in insects and mites. Comp Biochem Physiol, 64A: 191-200

Arlian LG and Veselica MM (1981a) Effect of temperature on the equilibrium body water mass in the mite Dermatophagoides farinae. Physiology Zoology, 54/4: 393-399

Arlian LG and Veselica MM (1981b) Re-evaluation of the humidity requirements of the house dust mite *Dermatophagoides farinae* (Acari: Pyroglyphidae). Journal of Medical Entomology, 18/4: 351-352

Arlian LG and Wharton GW (1974) Kinetics of active and passive components of water exchange between the air and a mite, *Dermatophagoides farinae*. Journal of Insect Physiology, 20: 1063-1077

Blythe, M.E (1976) Some aspects of the ecological study of the house dust mite. Brit. J. Dis. Chest 70: 3-31.

Boer R de (2000) Rates of water vapour uptake and loss by the house dust mite, Dermatophagoides pteronyssinus (Acari: Pyroglyphidae). Proceedings of Experimental & Applied Entomology, 11: 73-76

Boer R de and Kuller K (1997) Mattresses as a winter refuge for house-dust mite populations. Allergy, 52: 299-305

Boer R de, Kuller K and Kahl O (1998) Water balance of Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) maintained by brief daily spells of elevated air humidity. Journal of Medical Entomology, 35/6: 905-910

Bronswijk JE van (1981) House Dust Biology for Allergists, Acarologists and Mycologists. NIB Publishers, The Netherlands

Case T J (2000) An Illustrated Guide to Theoretical Ecology. Oxford Univ Press. New York.

Colloff, M. J (1987a) Effects of temperature and relative humidity on development times and mortality of eggs from laboratory and wild populations of the European house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae). Experimental and Applied Acarology 3 : 279-289.

Colloff, M. J (1987b) Differences in development time, mortality and water loss between eggs from laboratory and wild populations of *Dermatophagoides pteronyssinus*. Experimental and Applied Acarology 3 : 191-200.

Colloff M (1998) Taxonomy and identification of dust mites. Allergy 53 Supplement 48 : 7-12

Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S and Ridley I (2006). A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Experimental and Applied Acarology, 79(2): 127-148

Cunningham MJ (2000) A proposed experimental programme towards control of dust mites by microclimate modification, in Mites, Asthma and Domestic Design III (Eds: Siebers R, Cunningham M, Fitzharris P and Crane J). Wellington Asthma Research Group, Department of Medicine, Otago University, 2000

Dobson R.M (1979) Some effects of microclimate on the longevity and development of *Dermatophagoides pteronyssinus* (Trouessart). Acarologia 21: 482-486.

Ford AW, Rawle FC, Lind P, Spieksma FTM, Lowenstein H and Platts-Mills TAE (1985) Standardization of *Dermatophagoides pteronyssinus*: Assessment of potency and allergen content in ten coded extracts. International Archives of Allergy and Applied Immunology 76: 58-67.

Gamal-Eddin FM, Abou-Sinna FM, Tayel SE, Aboul-Atta AM, Seif AM, Gaafar SM (1983a). Duration of the developmental stages of house dust mites *Dermatophagoides farinae* and DP under controlled temperatures and relative humidities to pave the way in from of the workers in the field of house dust mite bronchial asthma. 1. Pre-imaginal period. Journal of the Egyptian Society of Parasitology 13 : 319-334.

Gamal-Eddin FM, Shehata KK, Tayel SE, Abou-Sinna FM, Aboul-Atta AM, Seif AI, Imam MH, Hafez AH (1983b). Duration and the development stages of house-dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled conditions to pave the way in front of the workers in the field of house-dust mite asthmatic bronchitis. 2. Oviposition period, fecundity and oval duration. Journal of the Egyptian Society of Parasitology, 13: 557-581

Gamal-Eddin FM, Shehata KK, Abou-Sinn FM, Tayel SE, Aboul-Atta AM, El-Ahmedaawy BA, Fayed MA, Hafez AH and Imam MH (1983c) Duration of the developmental stages of house dust mites DF and DP under controlled conditions to pave the way in from of the workers in the field of house dust mite asthmatic bronchitis 3. Larval Duration. Journal of the Egyptian Society of Parasitology 13 : 583 – 595

Hart, BJ. and Fain, A (1988) Morphological and biological studies of medically important house-dust mites. Acarologia 29: 285-295.

Ho, TM. and Nadchatram, M (1984) Life cycle and longevity of *Dermatophagoides pteronyssinus* (Trouessart) (Acarina: Astigmata: Pyroglyphidae) in a tropical laboratory. Trop. Biomed 1: 159-162.

Hodgson, R.K (1976) Sex ratio and sex determination in the American house dust mite, Dermatophagoides farinae. Annals Ent. Soc America, 69: 1085-6.

Nisbet, RM. and Gurney, WSC. (1982) Modelling Fluctuating Populations. John Wiley and Sons, Chichester.

Leslie, PH. (1945) On the use of matrices in certain population mathematics. Biometrika, 35: 183-212.

Pretlove S, Oreszczyn T, Ridley I, Wilkinson T and Crowther D (2005) A steady state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements. Building Services Engineering Research & Technology, 26/4: 301-314

Pike A, Cunningham M and Lester P (2005) Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) at constant and simultaneously fluctuating temperature and humidity conditions. Journal of Medical Entomology, 42/3: 266-269

Ridley I, Biddulph P, Oreszczyn T, Pretlove S, Crowther D, Wilkinson T, Ucci M and Hart B (2006) A model to predict the hygrothermal conditions experienced by house dust mites within beds. Submitted to Indoor Air.

Spieksma, F. Th M (1967) The House Dust Mite *Dermatophagoides pteronyssinus* (Trouessart, 1897). PhD Thesis, Univ. of Leiden, 65 pp.

U.S. Department of Energy (US-DOE) (2004) EnergyPlus Energy Simulation Software. http://www.eere.energy.gov/buildings/energyplus

Voorhorst R, Spieksma F and Varekamp H (1969) House Dust Atopy and the House Dust Mite, *Dermatophagoides pteronyssinus* (Trouessart, 1897). Stafleu's Scientific Publishing Company, Leiden

Wharton G (1978) Uptake of water vapour by mites and mechanisms utilized by the Acaridei. In Comparative Physiology: Water, Ions and Fluid Mechanics (Eds: Schmidt-Nielsen K, Bolis L and Maddrell S). Cambridge University Press, Cambridge.

Wilkinson T, Horwood J, Cox P, Crowther D, Ridley I, Pretlove S and Oreszczyn T (2002) Factors affecting the carrying capacity (K) of a mattress for the house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) 11th International Congress of Acarology. Universidad Nacional Autonoma de Mexico, Merida, Yucatan, Mexico, 8th-13th September 2002

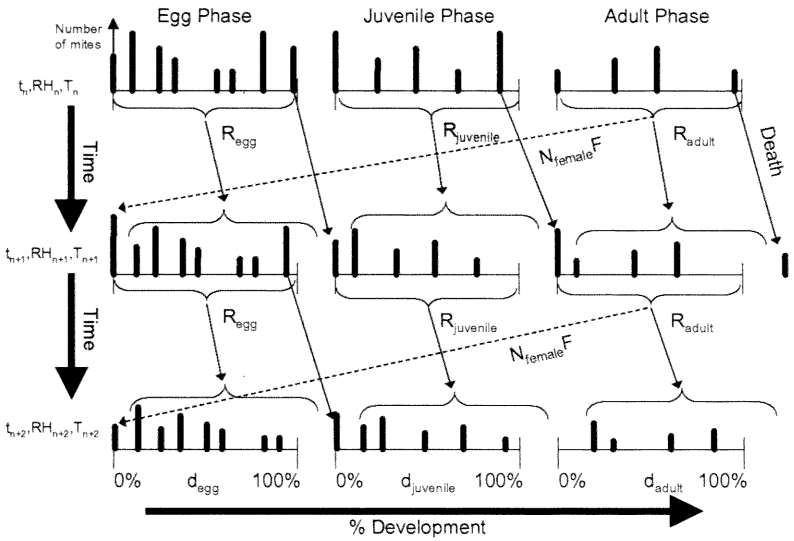

Figure 1 Schematic diagram of a simplified mite life cycle

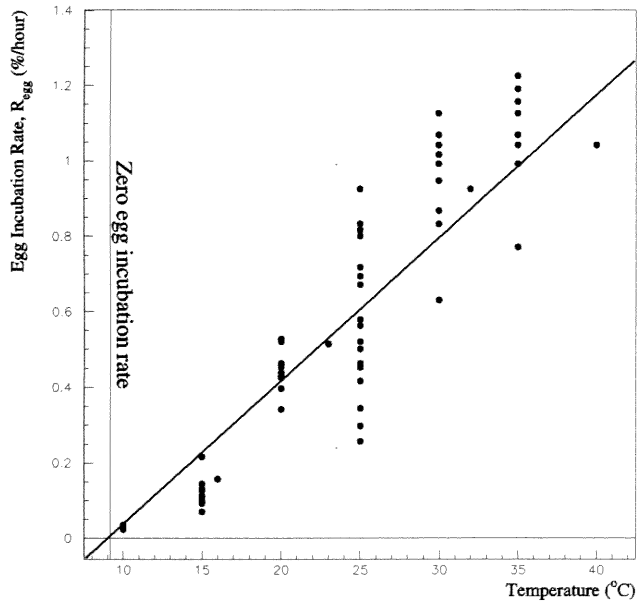

Figure 2 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite egg development rate as a function of temperature

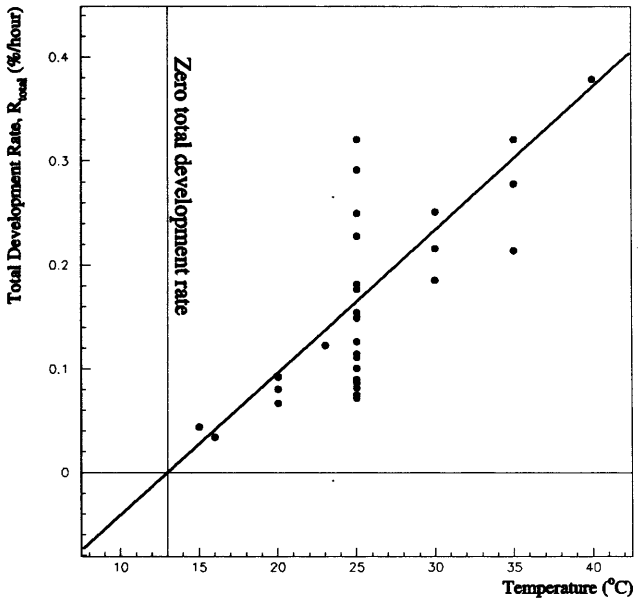

Figure 3 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite total development rate (from egg to adult) as a function of temperature

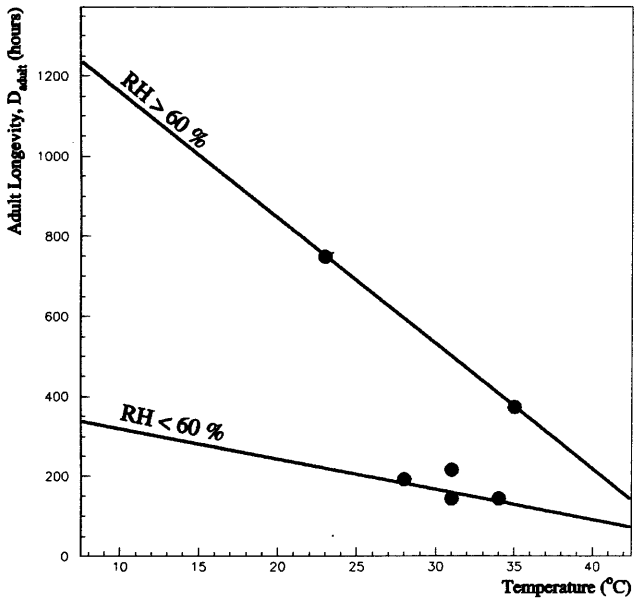

Figure 4 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite adult longevity as a function of temperature

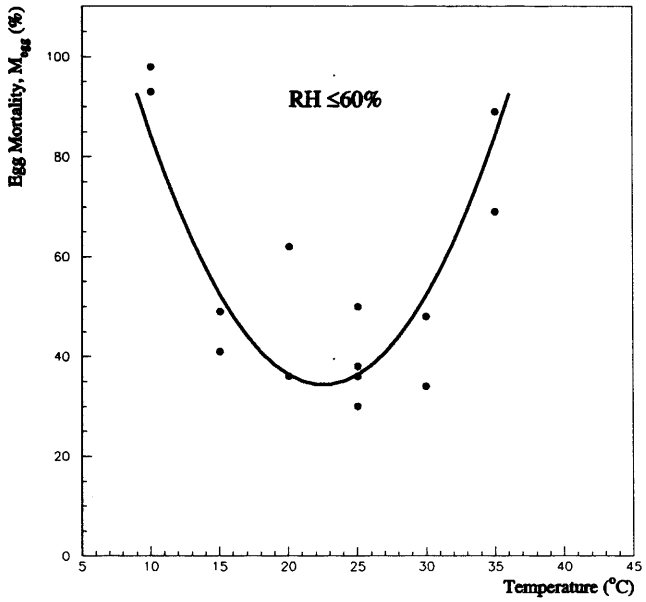

Figure 5 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite egg mortality as a function of temperature for relative humidity values less than 60%

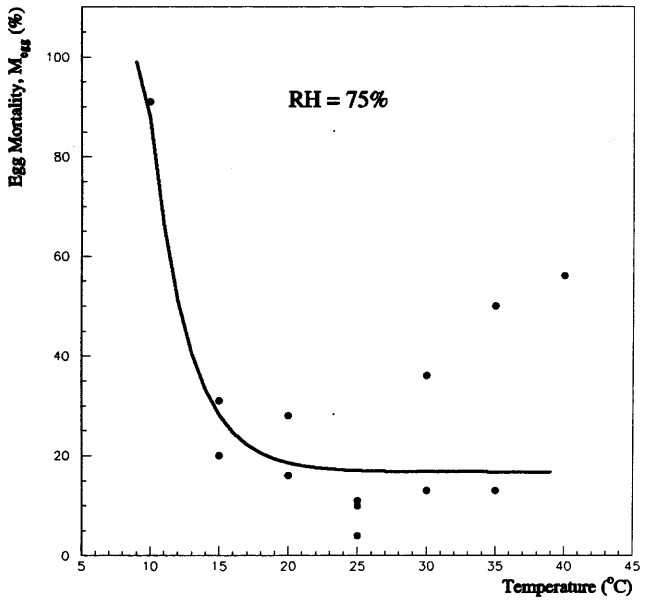

Figure 6 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite egg mortality as a function of temperature at a fixed relative humidity of 75%

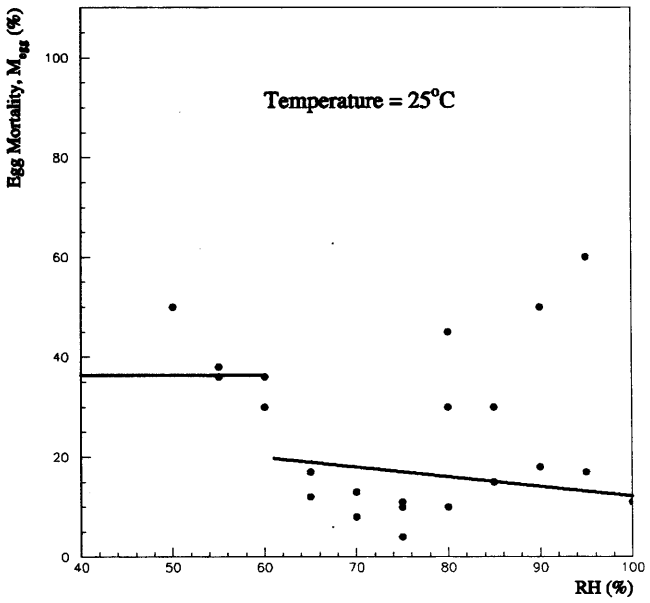

Figure 7 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite egg mortality as a function of relative humidity at a fixed temperature of 25°C

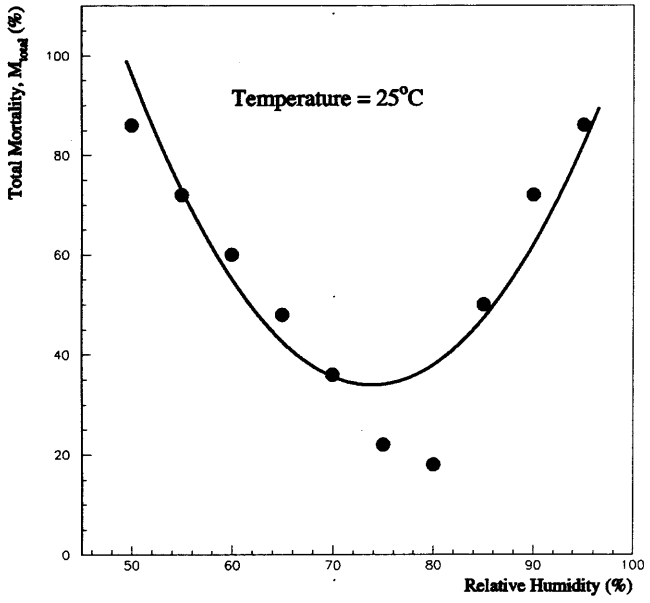

Figure 8 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite total mortality, from egg to adult, as a function of relative humidity for a fixed temperature of 25°C

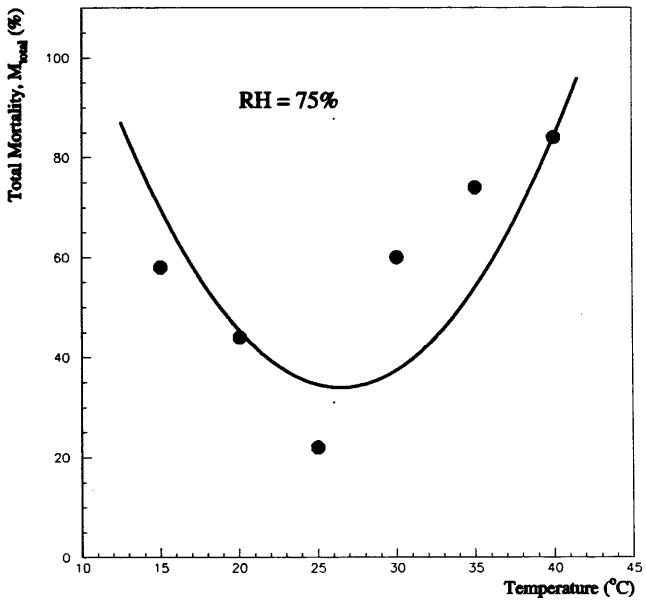

Figure 9 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite total mortality, from egg to adult, as a function of Temperature for a fixed relative humidity of 75%

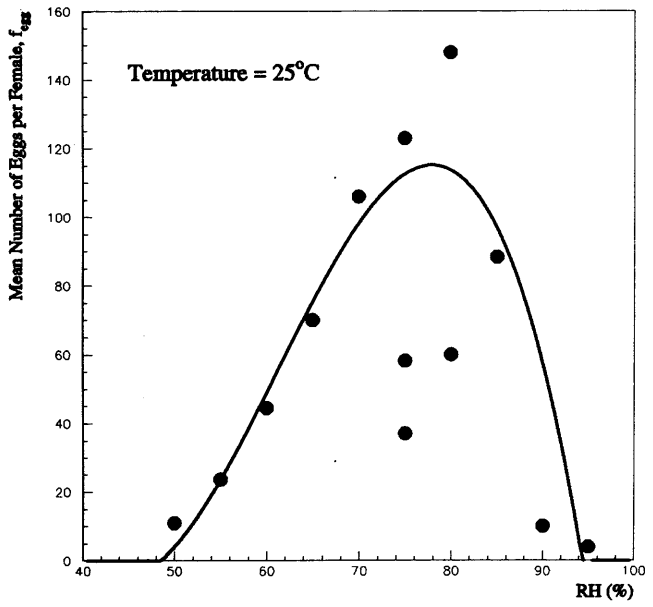

Figure 10 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite mean number of eggs per female during adult life as a function of relative humidity at a fixed temperature of 25°C

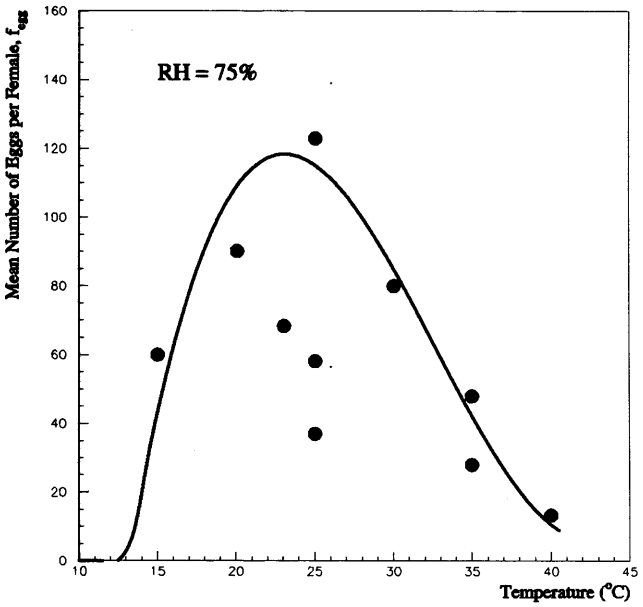

Figure 11 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite mean number of eggs per female during adult life as a function of Temperature at a fixed relative humidity of 75%

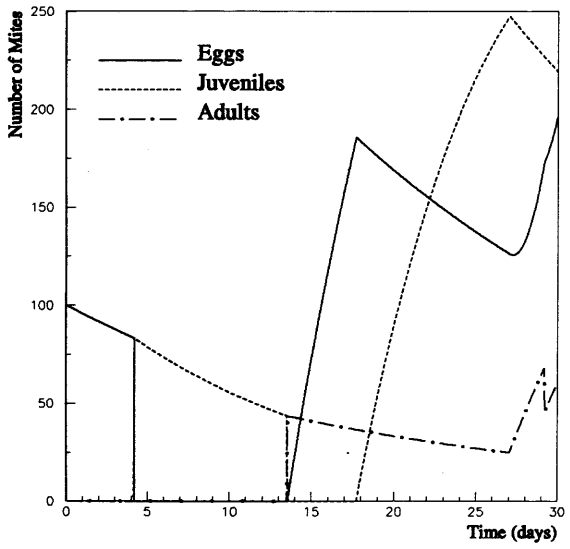

Figure 12 Prediction of the population dynamics of a batch of 100 freshly laid eggs held at a constant temperature of 35°C and a constant relative humidity of 75%. The total number of eggs in all batches is plotted as a solid line, the total number of juveniles as a dashed line and the total number of adults as a dash-dotted line

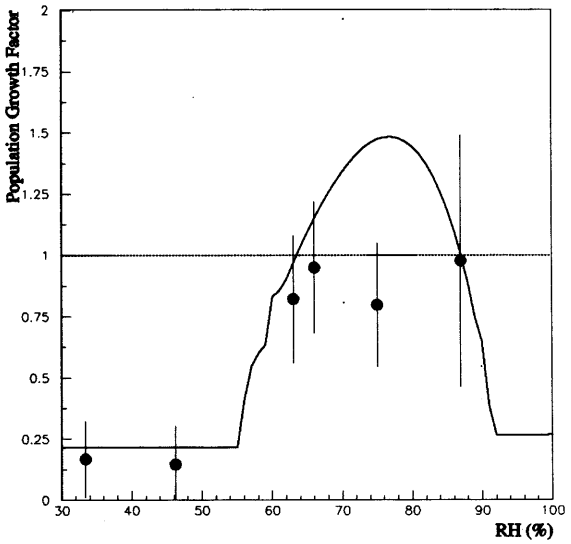

Figure 13 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) for a range of RH values at a fixed temperature of 15°C.

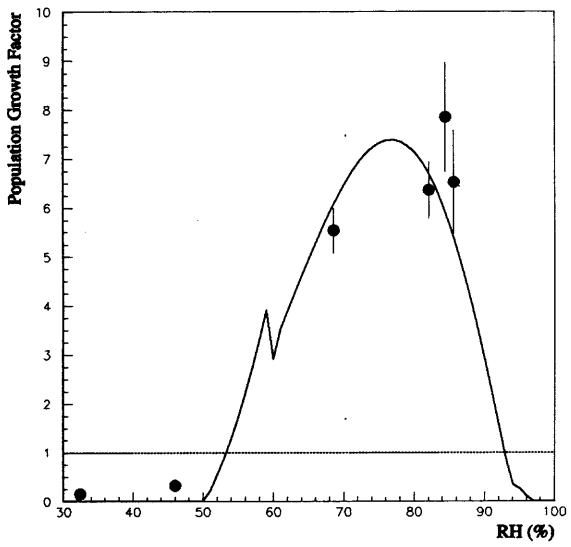

Figure 14 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae)for a range of RH values at a fixed temperature of 20°C.

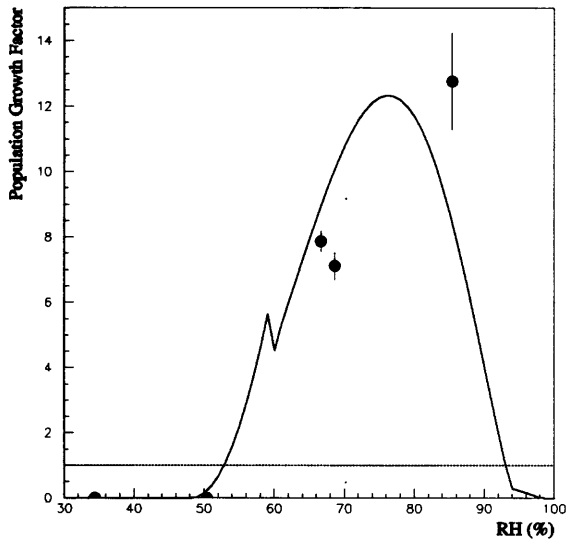

Figure 15 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae)for a range of RH values at a fixed temperature of 25°C.

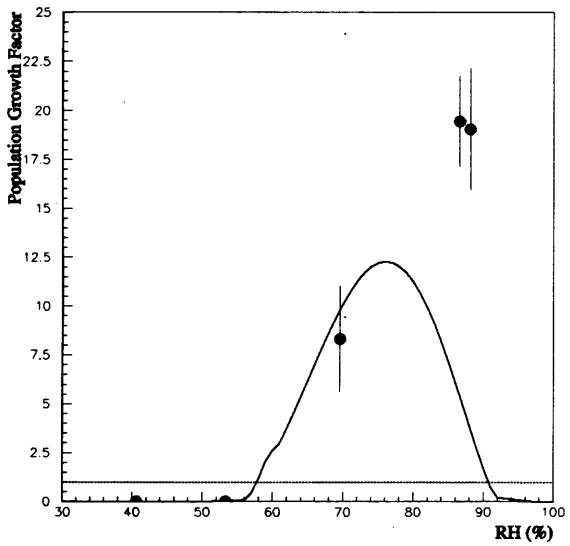

Figure 16 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae)for a range of RH values at a fixed temperature of 30°C.

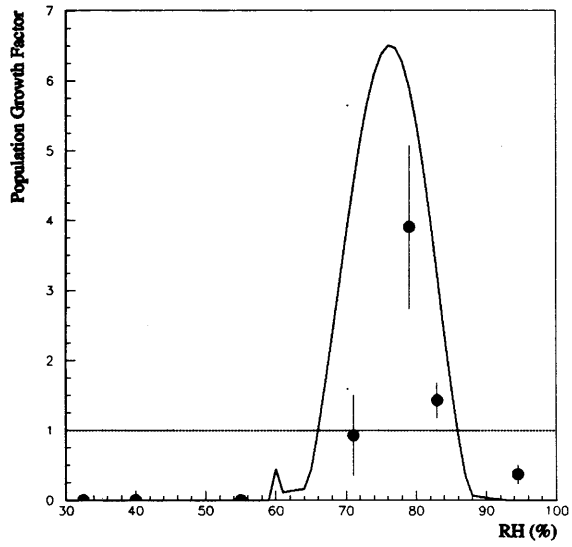

Figure 17 Comparison of the POPMITE prediction (solid line) with data from Crowther et al (2006) of the population growth multiplication factor of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae)for a range of RH values at a fixed temperature of 35°C.

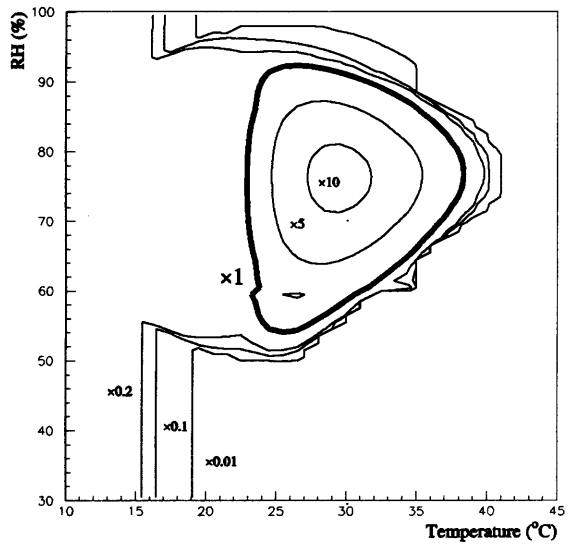

Figure 18 A contour plot of the complete mite population (eggs, juveniles and adults) growth factor after 30 days as predicted with the POPMITE model starting with 100 freshly laid eggs, for a range of relative humidity and temperature combinations

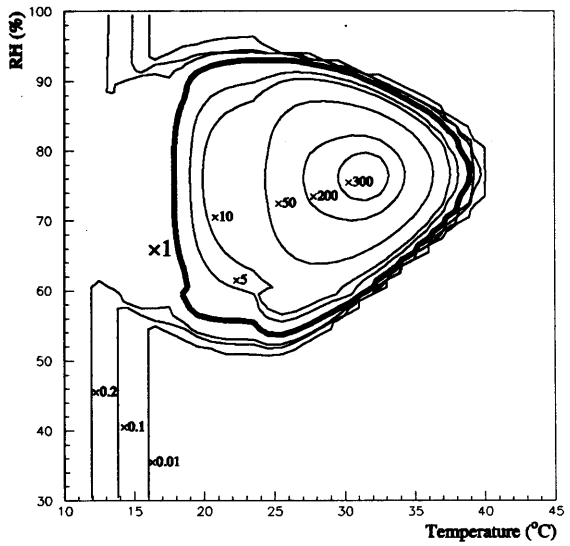

Figure 19 A contour plot of the complete mite population (eggs, juveniles and adults) growth factor after 60 days as predicted with the POPMITE model starting with 100 freshly laid eggs, for a range of relative humidity and temperature combinations

Table 1 Development duration for *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite eggs in days, [a]Colloff (1987a, 1987b), [b]Gamal Eddin et al. (1983b), [c]Arlian et al. (1990), [d]Spieksma (1967)

	Temperature(°C)																
RH(%)	10	15		16	20		23	25			30		32	35			40
50									16.2[b]								
55	180[a]	59[a]			9.7[a]			6.2[a]	14[b]		5[a]			4.2[a]			
60	155[a]	42[a]			9.2[a]			6[a]	12.1[b]		4.8[a]			4.2[a]			
65	120[a]	37[a]			9.1[a]			5.8[a]	10[b]		4.4[a]			3.9[a]			
70	135[a]	29[a]			7.9[a]			5.1[a]	9.2[b]		4.1[a]			4[a]			
75	145[a]	32[a]	19.3[b]	26.6[c]	7.9[a]	12.2[b]	8.1[c]	4.5[a]	8.3[b]		3.9[a]	6.6[b]	4.5[c]	3.9[a]	5.4[b]	3.9[c]	4[b]
80	150[a]	33[a]			8[a]			5[a]	7.2[b]	6[d]	3.7[a]			3.7[a]			
85	160[a]	38[a]			9[a]			5.2[a]	8[b]		4[a]			3.6[a]			
90	150[a]	42[a]			9.8[a]			9[a]	10[b]		4[a]			3.5[a]			
95	150[a]	44[a]			9.5[a]			5[a]	14[b]		4.1[a]			3.4[a]			
100	175[a]	45[a]			10.5[a]			5.2[a]			4.2[a]			3.9[a]			

Table 2 Total development time (days) of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mites from egg to adult, [a]Gamal Eddin et al (1983a), [b]Arlian et al. (1990) , [c]Arlian (1975), [d]Dobson (1979) , [e]Hart and Fain (1988), [f]Blythe (1976), [g]Anderson (1988), [h]Ho and Nadchatram (1984), [i]Spieksma (1967)

	Temperature(°C)															
RH(%)	15	16	20		23	25						30		35		40
50						58[a]										
55						51[a]										
60						46.5[a]										
65						41.5[a]										
70						37.5[a]										
75	95[a]	123[b]	62.4[c]	52[a]	34[b]	36.5[a]	14.3[c]	14.3[e]	33[f]	28[h]		19.3[b]	22.5[a]	15[b]	19.5[a]	11[a]
80			45.3[c]	45.2[d]		27[a]	18.3[c]	16.7[d]	13[d]	23.6[g]	23[i]	16.6[d]		13[d]		
85						36.5[a]										
90						48[a]										
95						56[a]										

Table 3 Adult longevity of *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mites (days), [a]Arlian (1975) and [b]Arlian et al. (1990).

	Temperature(°C)				
RH(%)	23	28	31	34	35
40		8[a]	6[a]	6[a]	
50			9[a]	6[a]	
75	31.2[b]				15.5[b]

Table 4 *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) Egg mortality % death at the end of the egg phase, [a]Colloff (1987a, 1987b), [b]Gamal Eddin et al (1983b), [c]Spieksma (1967)

	Temperature(°C)												
RH(%)	10	15		20		25			30		35		40
50								50[b]					
55	93[a]	49[a]		62[a]		36[a]		38[b]	48[a]		89[a]		
60	98[a]	41[a]		36[a]		36[a]		30[b]	34[a]		69[a]		
65	81[a]	37[a]		26[a]		17[a]		12[b]	15[a]		32[a]		
70	92[a]	35[a]		13[a]		13[a]		8[b]	10[a]		24[a]		
75	91[a]	31[a]	20[b]	28[a]	16[b]	11[a]	10[b]	4[b]	13[a]	36[b]	13[a]	50[b]	56[b]
80	90[a]	17[a]		14[a]		30[a]	45[c]	10[b]	26[a]		5[a]		
85	79[a]	21[a]		24[a]		15[a]		30[b]	8[a]		5[a]		
90	84[a]	18[a]		14[a]		18[a]		50[b]	16[a]		10[a]		
95	85[a]	23[a]		16[a]		17[a]		60[b]	14[a]		13[a]		
100	90[a]	26[a]		19[a]		11[a]			7[a]		15[a]		

Table 5 Total egg to adult mortality of the *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite, Gamal Eddin et al. (1983a)

	Temperature(°C)					
RH(%)	15	20	25	30	35	40
50			86			
55			72			
60			60			
65			48			
70			36			
75	58	44	22	60	74	84
80			18			
85			50			
90			72			
95			86			

Table 6 Mean number of eggs produced by a female *Dermatophagoides Pteronyssinus* (Acari: Pyroglyphidae) mite during her adult life, [a]Gamal Eddin et al. (1983b), [b]Arlian et al. (1990) , [c]Hart and Fain (1988), [d]Spieksma (1967).

	Temperature(°C)								
RH (%)	15	20	23	25		30	35		40
50				11[a]					
55				23.7[a]					
60				44.5[a]					
65				70[a]					
70				106[a]					
75	60[a]	90.2[a]	68.4[b]	123[a]	58.2[c]	80[a]	28[a]	48[b]	13.3[a]
80				148[a]	60[d]				
85				88.4[a]					
90				10[a]					
95				4[a]					

Appendix A.0: Published Papers

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A.0.3: *Reproduction and development of laboratory and wild house dust mites (Dermatophagoides pteronyssinus (Acari: Pyroglyphidae)) and their relation to the natural dust ecosystem*

B.J. Hart[1], D. Crowther[2], T. Wilkinson[2,3], P. Biddulph[2,4], **M. Ucci**[4], S. Pretlove[5], I. Ridley[4] And T. Oreszczyn[4]

[1]Royal Agricultural College, Stroud Road, Cirencester, Gloucestershire GL7 6JS, UK
[2]Martin Centre, Cambridge University, 1 Scroope Terrace, Cambridge CB2 1PX, UK
[3]Medical Entomology Centre, Barrington Road, Shepreth, Royston SG8 6QZ, UK
[4]Bartlett School of Graduate Studies, University College London WC1E 6BT, UK
[5]School of Architecture & Landscape, Kingston University, London KT1 2QJ, UK

Abstract

Life histories of 'wild' house dust mites (*Dermatophagoides pteronyssinus* (Trouessart)) were compared to laboratory cultures, using a diet consisting of skin and dust or a laboratory diet of dried liver and yeast. Under constant conditions of 25°C, 75% relative humidity (RH), fecundity and rate of reproduction were higher in laboratory cultures on both diets compared to wild mites. There were also trends for a shorter pre-reproductive period and more rapid egg development of laboratory mites compared to wild mites. Overall there was little effect of diet on either strain of mites at 75% RH. At low RH (64%), fecundity was significantly lower (for both strains on both diets) and there were also trends for longer pre-reproductive period, reduced rate of reproduction, reduced adult survival and prolonged egg/juvenile development compared to 75% RH. Additionally egg and juvenile mortality were significantly higher on the liver/yeast diet. Overall the skin/dust diet favoured both strains of mites at 64% RH. On the liver/yeast diet at 64% RH wild mite adults performed significantly better than laboratory mites and egg mortality was lower. These results suggest that laboratory mites have stronger reproduction and development than wild mites, except when under environmental stress, and that diet is a significant factor, particularly in sub-optimal conditions. This could have important implications for predictive models of house dust mite populations in their natural habitat. Ideally, such models should be developed using data from wild dust mite populations reared on a natural diet.

Key Words:

Dermatophagoides pteronyssinus, wild populations, life history.

INTRODUCTION

Population models to predict house dust mite populations in the home are currently under development (Pretlove *et al.* 2001, 2005; Crowther *et al.* 2006, Biddulph *et al.* 2007). Their aim is to assist in the effective control of mites by manipulating the temperature and relative humidity in their habitats, psychrometric conditions being known to play a crucial role in their survival (Cunningham 1999, Pretlove *et al.* 2002). The models set out to simulate, first, psychrometric conditions in mite habitats (given climate and building characteristics) and, second, the effect of these conditions on house dust mite populations. In this way the most successful and feasible strategies for achieving psychrometric control can be determined, whether by improving ventilation or by a combination of modifications to building design, building operation and occupant behaviour (e.g. with respect to moisture production, window opening habits, etc.). However, the population models upon which these simulations depend require mite physiology data inputs which relate to the house dust ecosystem.

Data on house dust mite reproduction and development have, until now, predominantly been obtained from mite cultures that have been reared for many years in laboratory conditions (Spieksma 1967, Blythe 1976, Dobson 1979, Gamal-Eddin *et al.* 1983a, 1983b, 1983c; Ho and Nadchatram 1984, Andersen 1988, Hart and Fain 1988, Arlian *et al.* 1990). However, in 1987, Colloff (1987a, 1987b) studied eggs from wild populations of house dust mites and suggested that they differed from eggs from laboratory populations with respect to their development time, mortality and water loss. There have been no subsequent studies on wild populations of house dust mites and no data are available on juvenile or adult physiology from wild cultures.

The principal aims of this study were therefore to obtain more detailed information on the physiology of wild house dust mite populations (*Dermatophagoides pteronyssinus* (Trouessart) compared to laboratory populations and to determine the importance of wild mite data for predictive mite

population models compared to existing data from long-term laboratory populations.

MATERIALS AND METHODS

Mite Cultures.

The laboratory strain of *D. pteronyssinus* had been reared for at least 10 years under constant laboratory conditions of 25°C temperature and 75% relative humidity (RH). Prior to experiments they were reared under these constant hygrothermal conditions on a typical optimised liver/yeast diet of ground dried porcine liver (Oxoid, UK) and brewers yeast (Holland and Barrett UK 1:1 (w:w)).

A 'wild' strain of *D. pteronyssinus* was collected from carpet dust from a UK home in September 2004. From the time of collection, this culture was reared under fluctuating hygrothermal conditions, that is to say fluctuating room temperatures and a diurnal RH fluctuation of 8h at 64% RH and 16h at 75% RH. Wild cultures were reared on a mixture of 1:0.1 (w:w) house dust and non-degreased (fresh) skin scales, with no addition of yeast. Experiments using these wild cultures were started in July 2005.

Mite Physiology Studies.

Glass micro-culture vials 12 mm diameter x 10 mm depth were used to hold individual couples (males attached to tritonymphs) isolated from the laboratory or wild cultures for determination of adult survival and reproduction. Glue was applied around the rim of the vials to prevent escape of the mites and an equal quantity of food was added to each vial. 10 couples were used for each assay and initially observations were made daily to determine pre-reproductive period and then 2-3 times weekly for further egg production and adult survival.

The liver/yeast diet (described above) and a skin/dust diet were used in separate experiments to determine the influence of diet on mite performance. To

standardise the skin/dust diet, a stock of mattress dust was collected from the beds of a total of 20 non-smokers and pooled. It was then frozen at –20°C for a minimum of one week to kill any mites, sieved through a 500 micron mesh and then kept at room temperature for at least one month before use in experiments. The aim of the latter step was to enable recovery of house dust fungi after the freezing step. A pooled stock of skin scales was obtained using beard shavings collected from electric razors of 8 volunteers and were left untreated at room temperature before adding to the dust stock at the start of each experiment to provide a 1:1 (w:w) mixture. No yeast was added to this 'natural' skin/dust diet.

To obtain eggs for development studies, thirty adult females were added to glass micro-culture vials as described above containing either the liver/yeast diet or the pooled skin/dust diet. They were left at 25°C, 75% RH until 50 eggs were laid, the females were then removed and the eggs placed into the relevant hygrothermal conditions for the experiment. Observations were made daily for egg hatching and 2-3 times weekly for juvenile mortality and development.

Constant hygrothermal conditions of 25°C and 75% RH or 64% RH were used in separate experiments to represent optimal laboratory rearing conditions and the lower RH typical of a domestic environment respectively. RH was controlled inside airtight plastic boxes using saturated inorganic salt solutions (Winston & Bates 1960) and was verified periodically throughout experiments using an RH meter.

Principal components analysis (PCA, Legendre and Legendre, 1998) was implemented to assess correlations between response variables, and to test for significant effects of predictor variables on the principal components, thus avoiding inflation of type 1 errors. Provided the PCA showed a response variable was significantly affected by the treatment, it was assessed individually using analysis of variance (ANOVA, Sokal and Rohlf, 1995). Data were log-transformed to meet the assumptions of parametric tests: examination of residuals and fitted values showed that transformation was adequate to remove heteroscedasticity and non-normality of error variance. Significance was assumed at the 5% level ($P = 0.05$), and a Gaussian error distribution was used.

RESULTS

Adults

In the PCA, the first two principal components had eigenvalues greater than one, and together captured 72% of the total variation in the response variables. Principal component 1 (PC1) was positively correlated with reproductive period, female survival, fecundity and male survival, while PC2 was positively correlated with pre-reproductive period and negatively correlated with reproductive rate. The pattern of high factor loadings on the same components suggests that the dependent variables are highly inter-correlated, and likely to show similar patterns among the ANOVAs.

The three-way interaction between strain, diet and RH was a significant predictor of PC1 ($F = 68.1$; $df = 7, 71$; $P < 0.001$). All three manipulated variables were significant predictors of PC2, and the interaction between strain and RH was also significant ($F = 19.8$; $df = 4, 74$; $P < 0.001$). This demonstrates that the effects detected within each life history trait below were real, and not artefacts of accepting random patterns as significant due to the number of separate tests done.

Fecundity.

The three-way interaction between strain, diet and RH was significant ($F = 130.9$; $df = 7, 72$; $P < 0.001$). Fecundity was always higher at 75% compared to 64% RH for all strain and diet combinations (Tables 1 and 2). This was particularly striking on the liver/yeast diet at 64% RH, where fecundity was up to 25 times lower than at 75% RH and up to 10 times lower than on the skin/dust diet at 64% RH.

At 75% RH (Table 1) there was no effect of diet on fecundity, but the laboratory strain of mites had significantly higher fecundity than the wild strain on both diets ($F = 15.6$; $df = 1, 38$; $P < 0.001$).

At 64% RH (Table 2) the interaction between strain and diet was significant ($F = 86.0$; $df = 3, 36$; $P < 0.001$). Both mite strains had higher fecundity on the

skin/dust diet than on the liver/yeast diet, but on the skin/dust diet the laboratory strain had the highest fecundity, while on the liver/yeast diet the wild strain had higher fecundity.

Pre-reproductive Period.

The three-way interaction between strain, diet and RH was significant ($F = 27.9$; $df = 7, 72$; $P < 0.001$). Pre-reproductive period (defined here as the period between mating of female tritonymphs with males and production of first eggs) was shorter at 75% RH than at 64% RH for every combination of strain/diet (Tables 1 and 2). High RH shortened pre-reproductive period to less than any group at low RH, except for wild mites on the skin/dust diet.

At 75% RH (Table 1) there was a significant interaction between strain and diet ($F = 27.2$; $df = 3, 36$; $P < 0.001$). On both diets the laboratory mites had a shorter pre-reproductive period than the wild mites. Wild mites had a significantly longer pre-reproductive period on the skin/dust diet compared to the liver/yeast diet ($F = 32.0$; $df = 1, 18$; $P < 0.001$), but no effect of diet was seen in laboratory mites.

At 64% RH (Table 2) there were no significant differences between strains, and in both strains the skin/dust diet resulted in significantly longer pre-reproductive periods compared to the liver/yeast diet ($F = 18.9$; $df = 1, 18$; $P < 0.001$).

Reproductive Period.

The three-way interaction between strain, diet and RH was significant ($F = 33.1$; $df = 7, 72$; $P = 0.008$). On the liver/yeast diet both strains of mites showed markedly shorter reproductive periods at 64% RH compared to 75% ($F = 68.4$; $df = 2, 36$; $P < 0.001$). There was no significant response to RH in wild mites on the skin/dust diet, but in the lab strain fed on the skin/dust diet reproductive period was significantly longer at 64% than at 75% RH ($F = 5.9$; $df = 1, 18$; $P = 0.026$) (Tables 1 and 2).

There was no influence of diet on reproductive period at 75% RH and the only significant difference between strains was seen on the liver/yeast diet where wild

mites had a longer mean reproductive period than laboratory mites ($F = 68.4$; $df = 2, 36$; $P < 0.001$) (Table 1).

At 64% RH (Table 2) the magnitude of the response to diet differed between the two strains of mites ($F = 35.8$; $df = 3, 36$; $P = 0.003$). While both strains of mites showed significantly shorter reproductive periods on the liver/yeast diet compared to the skin/dust diet, the response was greater in the laboratory strain. As found at 75% RH, wild mites had a longer mean reproductive period than laboratory mites on the liver/yeast diet. In contrast, on the skin/dust diet laboratory mites had a significantly longer reproductive period than wild mites.

Reproductive Rate.

Reproductive rate was higher at 75% RH than at 64% in both mite strains and higher in the laboratory strain than in the wild strain at both RH levels ($F = 27.0$; $df = 2, 77$; $P < 0.001$) (Tables 1 and 2). Diet did not affect reproductive rate.

Female Survival.

Female survival (the period from mating of female tritonymphs with males and death) of both mite strains decreased at 64% RH compared to 75% RH on the liver/yeast diet ($F = 57.8$; $df = 3, 36$; $P < 0.001$) and this decline in survival was more marked in the laboratory mite strain than in the wild strain. On the skin/dust diet female survival increased with RH in the wild strain, but decreased with increasing RH in the laboratory strain ($F = 5.0$; $df = 3, 37$; $P = 0.005$) (Tables 1 and 2).

Diet had no significant effect on female survival at 75% RH and the only difference between strains at this RH was found on the liver/yeast diet, where survival of wild females was greater than laboratory-reared females ($F = 57.8$; $df = 3, 36$; $P < 0.001$) (Table 1).

At 64% RH (Table 2) the two mite strains differed in the magnitude of their response to diet ($F = 71.9$; $df = 3, 36$; $p < 0.001$). While female survival of both mite strains was lower on the liver/yeast diet compared to the skin/dust diet, the laboratory strain demonstrated a much greater increase in survival on the skin/dust

diet than did the wild mites. Wild females on the liver/yeast diet had greater survival than laboratory mites on this diet, whereas the laboratory strain had much greater survival than the wild strain on the skin/dust diet.

Male Survival.

The three-way interaction between mite strain, diet and RH was a significant predictor of male survival ($F = 20.8$; $df = 7, 71$; $P < 0.001$). Survival of males (survival time of males of unknown age during experiments) of both mite strains decreased at 64% RH compared to 75% RH on the liver/yeast diet ($F = 125.3$; $df = 1, 37$; $P < 0.001$). On the skin/dust diet this effect was seen only in the wild mite strain ($F = 29.5$; $df = 3, 35$; $P < 0.001$) and was less marked than that seen on the liver/yeast diet (Tables 1 and 2).

At 75% RH (Table 1) there was no effect of mite strain on male survival, however wild males on the skin/dust diet had greater survival than those on the liver/yeast diet at this RH ($F = 29.5$; $df = 3, 35$; $P < 0.001$).

At 64% RH (Table 2) again diet was the only significant predictor of male survival, with males of both strains on the skin/dust diet living longer than those on the liver/yeast diet ($F = 125.3$; $df = 1, 37$; $P < 0.001$).

Immature Development.

Only the first principal component had an eigenvalue greater than one, and it explained 78.8% of the variation in egg, juvenile and total development. PC1 was positively correlated with all of these factors, and had high factor loadings of all, suggesting that the dependent variables are highly inter-correlated and likely to show similar patterns among the ANOVAs.

The significant interactions between strain/RH and strain/diet in predicting PC1 ($F = 85.6$; $df = 5, 255$; $P < 0.001$) show that strain, diet and RH are all significant predictors of immature development. This demonstrates that effects detected within each developmental trait were real, and not artefacts of accepting random patterns as significant due to the number of separate tests done.

Egg Development.

The three-way interaction between mite strain, diet and RH was a significant predictor of egg development times ($F = 91.1$; $df = 7, 333$; $P < 0.001$). Compared to 75% RH, egg mortality at 64% was 40% higher in laboratory mites on the liver/yeast diet. On this diet eggs of both strains of mites developed faster at 75% RH compared to 64% ($F = 111.6$; $df = 7, 333$; $P < 0.001$). However, on the skin/dust diet, while the laboratory strain showed more rapid egg development at 75% compared to 64%, egg development in the wild strain was inhibited at 75% RH ($F = 100.6$; $df = 7, 333$; $P < 0.001$) (Tables 1 and 2).

At 75% RH (Table 1) there was no effect of diet on egg development, but eggs from the laboratory strain developed more quickly than those laid by the wild strain on both diets ($F = 52.7$; $df = 1, 198$; $P < 0.001$).

At 64% RH both mite strain and diet influenced egg development (Table 2). Development times were quicker when the laboratory mite strain was on its accustomed diet (liver/yeast) compared to the skin/dust diet and also when the wild mites were on their accustomed diet (skin/dust) compared to the liver/yeast diet ($F = 194.3$; $df = 1, 198$; $P < 0.001$). Strain effects were seen on the liver/yeast diet where wild mite eggs had much slower development than eggs from the laboratory strain ($F = 111.6$; $df = 7, 333$; $P < 0.001$), whilst the reverse was true on the skin/dust diet.

Juvenile Development.

No juveniles of either strain of mites completed development on the liver/yeast diet at 64% RH, thus comparisons could be made between strain and RH on the skin/dust diet only. At 75% RH both strains had faster juvenile development than at 64% ($F = 70.4$; $df = 5, 294$; $P < 0.001$) (Tables 1 and 2).

At 75% RH, juvenile development responded differently to diet between strains ($F = 24.3$; $df = 3, 196$; $P < 0.001$). Each strain had faster development on the diet to which they were accustomed compared to the alternative diet (Table 1).

At 64% RH, on the skin/dust diet the wild mites had slower juvenile development than the laboratory strain on this diet ($F = 60.6$; $df = 3, 196$; $P < 0.001$) (Table 2).

Total Development.

There was a significant three-way interaction of mite strain, diet and RH in predicting total development time ($F = 72.3$; $df = 4, 295$; $P < 0.001$). Total development was faster at 75% RH than at 64% RH in both strains (Tables 1 and 2).

At 75% RH, in the laboratory mite strain the skin/dust diet markedly delayed total development compared to the liver/yeast diet, but no significant effect of diet was detected in the wild strain ($F = 12.0$; $df = 1, 198$; $P < 0.001$) (Table 1).

At 64% RH there were no significant differences in total development of the two mite strains on the skin/dust diet (Table 2).

Discussion

This study has provided life history parameters of laboratory-reared *D. pteronyssinus* on a laboratory diet at 25°C and 75% RH, which generally appear to have higher reproductive parameters and faster development than previous reports (Spieksma 1967, Blythe 1976, Dobson 1979, Gamal-Eddin *et al.* 1983a, 1983b, 1983c; Ho and Nadchatram 1984, Colloff 1987a, 1987b; Andersen, 1988, Hart and Fain 1988, Arlian *et al.* 1990). Differences between these results are likely to be due to differences in strain of mites and/or diet. We have demonstrated the importance of diet in this paper and we are also currently investigating the extent to which different strains of wild mites may vary in their life history parameters.

There have been few published studies on life history parameters of laboratory-reared *D. pteronyssinus* at 25°C and 64%RH. However, our laboratory mites appeared to perform less well at low RH than previous reports (Gamal-Eddin *et al.* 1983a, 1983b, 1983c; Colloff 1987a, 1987b). This may be due to the liver/yeast diet of desiccated liver and yeast, which appeared to be unsuitable for mite reproduction and development at low RH compared to the skin/dust diet (see discussion below).

This study has also provided the first comprehensive data set of adult reproduction and immature survival and development of wild *D. pteronyssinus* in optimal and sub-optimal rearing conditions. Previously only egg survival and development has been reported by Colloff (1987a, 1987b), who suggested that eggs of wild mites survive better and develop more quickly than those of laboratory mites when reared in cool, dry conditions of temperature and RH, whereas in warm, humid conditions the reverse is true. Our results also suggest that laboratory mites perform better (higher fecundity and rate of reproduction, shorter pre-reproductive period and faster egg development) than wild mites in optimum rearing conditions (75% RH), but in sub-optimum conditions (64% RH) laboratory-reared mites perform less well (lower fecundity, shorter reproductive period, reduced female survival and higher egg mortality) on the liver/yeast diet than wild mites. In contrast, on the skin/dust diet at low RH, fecundity, rate of reproduction, reproductive period and female survival of laboratory mites were higher than found in wild mites, but rearing the laboratory mites prior to the experiment on an optimised liver/yeast diet is likely to have had an influence on their subsequent egg production and survival on the skin/dust diet.

In some arthropods specific traits can be selected after as little as five generations (Navarro *et al.* 1985, Yano and Takafuji 2002, Young *et al.* 2003). Therefore, during the period between collection and the start of experiments, it is possible that our wild mite cultures may have in part adapted to laboratory culture conditions and thus represent an intermediate stage between wild and fully adapted long-term laboratory cultures. However this seems unlikely, since our wild cultures were reared in conditions relating very closely to those found in the home (diurnally fluctuating hygrothermal conditions) and on a natural diet consisting of only skin scales and house dust.

The poor performance of adults and immatures on the liver/yeast diet compared to the skin/dust diet at low RH was particularly striking. Most existing data on reproduction and development of house dust mites have been obtained from mites reared on laboratory diets which are highly nutritious and provide good population development in optimum hygrothermal conditions, but such diets may not provide an ideal substrate for mite survival at low RH. This could explain the sparse data on survival, reproduction and development of house dust mites reared

at RH below 75%. However, Saint Georges-Gridelet (1984) previously reported high population growth of *D. pteronyssinus* at 64% RH on diets high in lipids. In this paper it was suggested that lipids present in skin scales in the house dust substrate could explain the survival of mites in their natural habitat where hygrometric conditions are often below the critical equilibrium activity (Arlian 1975, Arlian and Veselica 1981) of the mites. Our results appear to agree with this and suggest that data on mite performance on laboratory yeast-based diets, particularly in sub-optimal conditions, are unlikely to represent performance on a skin-based diet in their natural dust habitat.

Population models to predict dust mite populations in homes are currently under development using previously published data primarily from laboratory populations of mites reared on laboratory diets (Pretlove *et al.* 2001, 2005; Crowther *et al.* 2006, Biddulph *et al.* 2007). The present study has highlighted the requirement for a more comprehensive data set from wild mite populations reared on a natural diet for use in these models. Work is underway by the current authors to provide this data.

Another critical factor likely to have an influence on the life history parameters of house dust mites is fluctuating temperature and RH. De Boer *et al.* (1998) have shown that *D. pteronyssinus* can survive and produce eggs when held at low RH and given as little as 3 hours moist air per day. Arlian *et al.* (1999) suggested that the development of *D. farinae* (Hughes) was slower in fluctuating conditions of RH compared to constant high RH. More recently Pike *et al.* (2005) found that the population dynamics for *D. pteronyssinus* were similar in both fluctuating and constant conditions of temperature and RH. However, Colloff (1987a) proposed that laboratory mite populations were less able to withstand diurnal fluctuations in microclimate than wild populations of mites. This is also the subject of a future paper by the current authors using wild mites reared on a natural diet.

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References Cited

Andersen, A. 1988. Population growth and developmental stages of the house dust mite, *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). J. Med. Entomol. 25: 370-373.

Arlian, L.G. 1975. Dehydration and survival of the European house dust mite, *Dermatophagoides pteronyssinus*. J. Med. Entomol. 12: 437-442.

Arlian, L.G., J.S. Neal, and D. Vyszenski-Moher. 1999. Fluctuating hydrating and dehydrating relative humidities effects on the life cycle of *Dermatophagoides farinae* (Acari: Pyroglyphidae). J. Med. Entomol. 36: 457-461.

Arlian, L.G., C.M. Rapp, and S.G. Ahmed. 1990. Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). J. Med. Entomol. 27: 1035-1040.

Arlian, L.G., and M.M. Veselica. 1981. Re-evaluation of the humidity requirements of the house dust mite *Dermatophagoides farinae* (Acari: Pyroglyphidae). J. Med. Entomol. 18: 351-352.

Biddulph, P., D. Crowther, B. Leung, T. Wilkinson, B. Hart, T. Oreszczyn, S. Pretlove, I. Ridley, and M. Ucci. 2007. Predicting the population dynamics of the house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) in response to a constant hygrothermal environment using a model of the mite life cycle. Exp. Appl. Acarol. (in press).

Blythe, M.E. 1976. Some aspects of the ecological study of the house dust mite. Brit. J. Dis. Chest 70: 3-31.

Boer, R., K. de Kuller, and O. Kahl. 1998. Water balance of *Dermatophagoides pteronyssinus* (Acari : Pyroglyphidae) maintained by brief daily spells of elevated air humidity. J. Med. Entomol. 35: 905-910.

Colloff, M.J. 1987a. Differences in development time, mortality and water loss between eggs from laboratory and wild populations of *Dermatophagoides pteronyssinus*. Exp. Appl. Acarol. 3: 191-200.

Colloff, M.J. 1987b. Effects of temperature and relative humidity on development times and mortality of eggs from laboratory and wild populations of the European house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Exp. Appl. Acarol. 3: 279-289.

Crowther, D., T. Wilkinson, P. Biddulph, T. Oreszczyn, S. Pretlove, and I. Ridley. 2006. A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae). Exp. Appl. Acarol. 79: 127-148.

Cunningham, M.J. 1999. Modelling of some dwelling internal microclimates. Building and Environment 34: 523-536.

Dobson, R.M. 1979. Some effects of microclimate on the survival and development of *Dermatophagoides pteronyssinus* (Trouessart). Acarologia 2: 482-486.

Gamal-Eddin, F. M., F.M. Abou-Sinna, S.E. Tayel, A.M. Aboul-Atta, A.M. Seif. and S.M. Gaafar. 1983a. Duration of the developmental stages of house dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled temperatures and relative humidities to pave the way in front of the workers in the field of house dust mite asthmatic bronchitis. 1. Pre-imaginal period. J. Egypt. Soc. Parasitol. 13: 319-334.

Gamal-Eddin, F.M., K.K. Shehata, F. Abou-Sinna, S. Tayel, A.M. Aboul-Atta, B.A. El-Ahmedaawy, M.A. Fayed, A.H. Hafez, and M. Imam. 1983b. Duration of the development stages of house-dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled conditions to pave the way in front of the workers in the field of house-dust mite asthmatic bronchitis. 2. Oviposition period, fecundity and oval duration. J. Egypt. Soc. Parasitol. 13: 557-581.

Gamal-Eddin, F.M., K.K. Shehata, F.M. Abou-Sinna, S.E. Tayel, A.M. Aboul-Atta, B.A. El-Ahmedaawy, M.A. Fayed, A.H. Hafez, and M. Imam, M. 1983c. Duration of the developmental stages of house dust mites *Dermatophagoides farinae* and *D. pteronyssinus* under controlled conditions to pave the way in front of the workers in the field of house dust mite asthmatic bronchitis 3. Larval Duration. J. Egypt. Soc. of Parasitol. 13: 583–595.

Hart, B.J., and A. Fain. 1988. Morphological and biological studies of medically important house-dust mites. Acarologia 29: 285-295.

Ho, T.M., and M. Nadchatram. 1984. Life cycle and survival of *Dermatophagoides pteronyssinus* (Trouessart) (Acarina: Astigmata: Pyroglyphidae) in a tropical laboratory. Trop. Biomed. 1: 159-162.

Legendre, P. and L. Legendre. 1998. Numerical Ecology. 2nd ed. Elsevier Publ, The Netherlands.

Navarro, S., R. Dias, and E. Donahaye. 1985. Induced tolerance of *Sitophilus oryzae* adults to carbon dioxide. J. Stored Products Res. 21: 207-213.

Pike, A.J., M.J. Cunningham, and P.J. Lester. 2005. Development of *Dermatophagoides pteronyssinus* (Acari: Pyroglyphidae) at constant and simultaneously fluctuating temperature and humidity conditions. J. Med. Entomol. 42: 266-269.

Pretlove, S., I. Ridley, J. Horwood, B. Leung, P. Cox, D. Thomson, N. Baker, D. Crowther, and T. Oreszczyn. 2001. A combined transient hygrothermal and population model of house dust mites in beds. Proceedings of IAQ 2001: Moisture, Microbes and Health Effects: Indoor Air Quality and Moisture in Buildings, ASHRAE, San Francisco, USA, November 4 – 7, 2001.

Pretlove, S., R. Critchley, R. Howard, and T. Oreszczyn. 2002. The Nottingham energy, health and housing study: reducing relative humidity, dust mites and asthma. Building Services Engineering Research & Technology 23: 43-55.

Pretlove, S., T. Oreszczyn, I. Ridley, T. Wilkinson, and D. Crowther. 2005. A steady state model for predicting hygrothermal conditions in beds in relation to house dust mite requirements. Building Services Engineering Research & Technology 26 (4): 301-314.

Saint Georges-Gridelet, D. de 1984. Effects of dietary lipids on the population growth of *Dermatophagoides pteronyssinus*. Acarology 6: 351-357.

Sokal, R.R., and F.J. Rohlf. 1995. Biometry. WH Freeman Publ., USA.

Spieksma, F.Th.M. 1967. The House Dust Mite *Dermatophagoides pteronyssinus* (Trouessart, 1897). PhD Thesis, Univ. of Leiden, 65 pp.

Winston, P.W., and D.H. Bates. 1960. Saturated solutions for the control of humidity in biological research. Ecology 41: 232-237.

Yano, S., and A. Takafuji. 2002. Variation in the life history pattern on *Tetranychus urticae* (Acari: Tetranychidae) after selection for dispersal. Exp. Appl. Acarol. 27: 1-10.

Young, H.P., D.W. Bailey, and R.M.R.M. Roe. 2003. Spinosad selection of a laboratory strain of the tobacco budworm, *Heliothis virescens* (Lepidoptera: Noctuidae), and characterization of resistance. Crop Protection 22: 265-273.

Table 1. Life history parameters of laboratory and wild populations of *D. pteronyssinus* (DP) reared on liver/yeast (lab) and skin/dust (dust) diets at 25ūC and 75% RH.

	25ūC, 75% RH			
	Lab DP Lab diet	Wild DP Lab diet	Lab DP Dust diet	Wild DP Dust diet
ADULTS (n=10)				
Total fecundity per female	100 ± 33.4	66.9 ± 15.9	79.7 ± 12.6	60.9 ± 15.9
Pre-reproductive period (days)	2.2 ± 0.6	2.9 ± 1.1	2.6 ± 0.9	8.3 ± 3.4
Reproductive period (days)	35.5 ± 12.9	43.1 ± 22.9	27.0 ± 8.2	35.9 ± 18.9
Rate of reproduction (eggs/female/day)	3.0 ± 1.1	1.7 ± 0.5	3.2 ± 1.0	1.9 ± 0.8
Female survival (days)	45.2 ± 13.8	52.8 ± 22.1	39.9 ± 17.5	48.6 ± 20.2
Male survival (days)	34.8 ± 14.4	31.2 ± 5.9	33.9 ± 21.1	45.4 ± 16.1
IMMATURES (N=50)				
% egg mortality	0	0	0	0
Egg development time (days)	3.5 ± 0.9	4.5 ± 0.8	3.3 ± 0.5	5.4 ± 2.8
% juvenile mortality	0	0	0	0
Juvenile development time (days)	9.8 ± 1.2	10.9 ± 1.7	13.1 ± 0.7	10.1 ± 3.5
Total egg-adult development time (days)	13.3 ± 1.4	15.4 ± 2.4	16.4 ± 0.7	15.4 ± 4.4

Results show Mean ± SD.

Table 2. Life history parameters of laboratory and wild populations of *D. pteronyssinus* (DP) reared on liver/yeast (lab) and skin/dust (dust) diets at 25ūC and 64% RH.

	25ūC, 64% RH			
	Lab DP Lab diet	Wild DP Lab diet	Lab DP Dust diet	Wild DP Dust diet
ADULTS (n=10)				
Total fecundity per female	3.8 ± 0.9	6.9 ± 3.2	48.0 ± 10.3	24.4 ± 5.5
Pre-reproductive period (days)	6.2 ± 1.9	6.0 ± 1.6	12.4 ± 6.2	10.1 ± 3.7
Reproductive period (days)	2.8 ± 1.9	6.3 ± 3.5	36.2 ± 8.8	29.4 ± 10.3
Rate of reproduction (eggs/female/day)	2.3 ± 1.7	1.3 ± 0.7	1.4 ± 0.6	0.8 ± 0.4
Female survival (days)	10.2 ± 2.4	18.7 ± 4.8	56.6 ± 13.1	33.1 ± 7.8
Male survival (days)	9.8 ± 2.9	11.6 ± 4.0	44.0 ± 14.1	37.9 ± 12.8
IMMATURES (N=50)				
% egg mortality	40	0	0	0
Egg development time (days)	4.5 ± 1.1	8.0 ± 0	8.0 ± 0	2.3 ± 1.0
% juvenile mortality	100	100	0	0
Juvenile development time (days)			16.0 ± 3.5	23.9 ± 9.8
Total egg-adult development time (days)			24.0 ± 3.5	26.0 ± 9.9

Results show Mean ± SD.

Appendix A.0: Published Papers

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A.0.4: *The psychrometric control of house dust mites: a pilot study*

M Ucci[a], SEC Pretlove[b], P Biddulph[c], T Oreszczyn[a], T Wilkinson[c,d] D Crowther[c] G Scadding[e], B. Hart[f], D Mumovic[a]

[a]The Bartlett School of Graduate Studies, University College London, London, UK; [b]Kingston University, School of Architecture & Landscape, London, UK; [c]The Martin Centre, Department of Architecture, University of Cambridge, Cambridge, UK; [d]The Medical Entomology Centre, Shepreth, Royston, UK; [e]Royal National Throat Nose and Ear Hospital, London, UK; [f]The Royal Agricultural College, Cirencester, UK.

Abstract

This paper describes a pilot intervention study on the effectiveness of house dust mite allergen avoidance for twelve asthmatic children (two being controls). In addition to mite allergen removal, the study included tailored advice aimed at reducing mite population growth via changes in moisture production, heating and ventilation habits. This paper focuses on the effects of this advice on household behaviour, hygrothermal conditions and mite populations. The efficacy of monitoring and modelling techniques is also discussed. The study highlighted a number of interrelated confounding factors which have to be addressed in future similar larger scale studies, but the results are promising with regards to the effectiveness of such studies.

Practical application

This study suggests that in temperate climates tailored advice on moisture production, heating and ventilation habits can lead to valuable changes in hygrothermal conditions, which in turn can result in reduced mite populations. However, pre-existing adverse building conditions may hinder such changes, and the effectiveness of tailored advice and of hygrothermal modifications is often difficult to assess. It is therefore recommended that any similar larger intervention study measures ventilation rates and adequately controls for a number of confounding variables - including the effect of changes in outdoor conditions and of the removal of existing mite populations. In this respect, hygrothermal population models can play a very useful role in the assessment of study effectiveness.

1.0 Introduction

House dust mites (HDM) can be found in beds, carpets and soft furnishings. Exposure to them can lead to allergic sensitisation and to rhinitis and asthma

symptoms. Noticeable differences have been found in the prevalences of atopy and asthma symptoms worldwide, with the UK having some of the highest values[1].

House dust mites absorb moisture from the air. If the ambient RH is too low, mites dehydrate and eventually die. This critical low RH is often referred to as the *Critical Equilibrium Humidity* (CEH), which is probably temperature-dependent for *Dermatophagoides pteronyssinus* (DP), the most common species in the UK[2]. Temperature also plays an important independent role in mite physiology, for example affecting egg-to-adult development times. Thus, by adequately controlling the hygrothermal conditions of mite microclimates (psychrometric control)[3], it should be possible to reduce mite populations. Because of the dependency of mite populations on hygrothermal conditions, their growth in temperate climates is usually greatest in late summer/early autumn, and least in the winter months, which are a crucial time for reducing mite populations[2]. For typical indoor temperatures, maintaining the average daily indoor RH below 50% is often recommended to reduce mite levels and their allergens. HDM can survive when exposed to brief spells of high RH, even when the daily average RH is below critical levels[4,5]. Nonetheless, the psychrometric control method is still viable, as mite development rates are much slower under such circumstances[4].

Most studies on the psychrometric control of house dust mites in housing have focused on mechanical ventilation. There is however scope for modifying residential hygrothermal conditions by changing heating and ventilation habits. For example, a UK study found that extractor fans in the kitchen were associated with lower HDM allergen concentrations[6]. Nonetheless, few intervention studies have attempted to reduce HDM levels through modifications of occupant behaviour alone. In winter 2005 the authors took part in a pilot study on the effectiveness of dust mite allergen avoidance for 12 asthmatic children. It was filmed by *Twenty Twenty Television*[7] and resulted in two 50-minutes episodes of the UK TV series 'Dispatches' on Channel 4 (April 2006). Due to its short time-scale and small sample size, the study did not aim to establish the clinical efficacy

of allergen avoidance, but to illustrate potential benefits and give researchers the opportunity to test a protocol for a larger future study. As well as the removal of mite and pet allergens, it included tailored advice aimed at reducing mite population growth via changes in moisture production, heating and ventilation habits. The study addressed four issues: 1) the effect of allergen removal on the children's health; 2) the effect of tailored advice on occupant behaviour and the resultant hygrothermal conditions; 3) the effect of the hygrothermal changes on mite populations; and 4) the efficacy of monitoring/modelling techniques. This paper will focus on the last three issues, however the results did reveal a (weak) correlation between health improvements and HDM allergen reduction. This is encouraging, particularly considering the limited statistical power of this study. This paper illustrates the methods and findings of the pilot study, with a view to discussing their implications for a future larger scale study.

2.0 Methodology

In October 2005, twelve asthmatic mite-sensitive children aged 6 to14 were selected in the London area by the TV production team[7]. Eleven dwellings were examined overall, since two of the children were siblings living in 1 dwelling (here termed bedroom/child 12a and 12b). The properties included: 4 flats, 1 detached house and 6 terraced houses. A pre-intervention analysis was carried out in November 2005, where baseline measurements were taken of: the children's health status; HDM numbers and allergen levels in each dwelling (child bedroom: mattress, pillow, one soft toy and floor; living room: sofa and floor - all using a standard protocol); hygrothermal conditions (monitored for 2 weeks, logging every 15 minutes); building characteristics (including airtightness via a fan-pressurisation test); and heating and ventilation habits. The fan-pressurisation results at 50 Pa were converted to an estimated air-infiltration rate in air changes per hour under average external conditions[8]. The children's asthma and health status was assessed by Dr Glenis Scadding, consultant physician at the Royal National Throat Nose & Ear Hospital. After the pre-intervention study, a number of interventions were carried out, followed by a post-intervention study, where the

children's health and the dwellings' hygrothermal conditions were monitored for 6 weeks (Dec 05-Jan 06).

After the baseline measurements, the following interventions were carried out: professional steam-cleaning of the child's bedroom and thorough cleaning of the dwelling (followed by further dust sampling); replacement of carpets in the child's bedroom with laminate flooring; covering mattresses, pillows and duvets with micro-porous mite-proof barriers; removing pets and cuddly toys; and avoiding exposure to environmental tobacco smoke. The participants were also advised to implement a thorough cleaning regime throughout the post-intervention period. Following the analysis of the pre-intervention study results, tailored advice was also provided on moisture production, heating and ventilation. Outdoor hygrothermal conditions were also monitored throughout the study. For the two households acting as controls, the interventions were carried out *at the end* of the post-intervention period, but their dwelling's hygrothermal conditions were monitored throughout the study. At the end of the study, further dust samples were taken, and a final medical examination was carried out.

3.0 Pre Intervention Study: Baseline Measurements and Hygrothermal Advice

Table 1 shows the baseline results. Since little dust was found in toys and pillows, it was concluded that allergen concentrations can be misleading at times. Therefore, the results were also expressed in terms of 'allergen load': total allergen weight collected for a given vacuumed area, corresponding to μg Der p1/m^2 (μg Der p1/total object area, for pillows and toys). The daily moisture production (kg/day) was estimated by using the questionnaire results and the moisture algorithm of Condensation Targeter II[9]. The Critical Equilibrium Humidity (CEH) was calculated as a function of temperature using DF data, which is the fullest dataset currently available[10].

Based on the pre-intervention study results illustrated in Table 1, tailored advice was provided to each household on the most appropriate heating, ventilation and moisture-production patterns, which could reduce house dust mite populations. Depending on the dwelling and occupant behaviour characteristics, each household was advised to implement one, or a combination of, the following measures: a) reducing moisture production; b) increasing ventilation levels; and c) increasing temperature levels.

For example, Household 1 – with low air infiltration rates, and highest RH and VPX levels – was advised to: 1) only dry clothes indoors in a well ventilated room, which is closed to the rest of the home; 2) use the extract fans in the kitchen and the bathroom during use, and for at least 15 minutes afterwards; 3) keep the trickle vents always open; and 4) leave the windows slightly open, for as long as possible. On the other hand, Household 11 - with low temperatures and high infiltration rates - was advised to increase indoor temperatures. Control households (6 and 8) did not receive the advice until the end of the study.

4.0 Post-intervention results

In all dwellings, the bedroom RH decreased from pre to post intervention periods, and the percentage of time the bedroom RH was greater than CEH ('% time RH>CEH') also decreased (Table 2). However, the reduction in indoor RH levels was partly due to changes in outdoor conditions. Although the average outdoor RH increased during the post-intervention, it was colder and the outdoor absolute humidity was lower. In order to disentangle the weather effect from the advice implementation, the pre and post intervention RHs were adjusted for each bedroom. It was assumed that if the pre and post intervention period had exactly the same weather conditions, the indoor temperatures would be the same, and the indoor RH would be dependent on the dwelling's vapour pressure excess, as well as on the outdoor vapour pressure. Since the outdoor conditions had been monitored for a longer period during the post intervention period, the indoor RHs were adjusted in relation to the post intervention weather conditions. The adjusted vapour pressure was calculated as follows:

$$Pre\_VP^{Adj} = Out\_VP + Pre\_VPX \quad [1]$$

$$Post\_VP^{Adj.} = Out\_VP + Post\_VPX \quad [2]$$

where Out_VP is the outdoor vapour pressure monitored during the post intervention study; Pre_VPX is the average vapour pressure excess measured during the pre intervention period; Post_VPX is the average vapour pressure excess measured during the post intervention period. Figure 1 illustrates a schematic representation of the adjustment calculation for the vapour pressure. The adjusted RH was then calculated as follows:

$$Pre\_RH^{Adj} = RH(Post\_T, Pre\_VP^{Adj}) \quad [3]$$

$$Post\_RH^{Adj} = RH(Post\_T, Post\_VP^{Adj}) \quad [4]$$

where Pre_RH^{Adj} is the adjusted pre intervention RH, as a function of the monitored post temperature (Post_T), and the adjusted pre vapour pressure (Pre_VP^{Adj}). The $Post\_RH^{Adj}$ is the adjusted post intervention RH, as a function of the monitored post temperature (Post_T), and the adjusted post vapour pressure ($Post\_VP^{Adj}$).

Once the impact of changes in outdoor conditions was taken into account, it was found that the reduction in bedroom RHs was smaller than the measured results (Figure 2) - particularly for some bedrooms (measured pre-post average RH difference: 12.1%; adjusted pre-post average RH difference: 5.1%, excluding control bedrooms). A paired t-test showed that there was a statistically significant difference ($p<0.01$) between pre and post bedroom RHs, for both the *measured* and the *adjusted* RH results. The importance of the adjustment procedure is highlighted by the case of the control Dwelling 8, where the measured bedroom RH decreased from the pre to the post intervention period. However, its adjusted RH *increased* (by a small amount, Fig 2). This was due to an increase in the measured post-intervention vapour pressure excess (Table 2), probably because of a reduction in window opening for the colder outdoor temperatures. It should also be highlighted that the other control dwelling (Dwelling 6) experienced an above average reduction in adjusted RH levels. However it is possible that Household 6 learnt about the advice provided to other families. It is also possible that the RH adjustment method described earlier may underestimate reductions in relative

humidity, since the VPX in dwellings can be dependent on outdoor conditions, with higher VPXs during cold weather - probably because occupants ventilate less when it is cold outside[11]. Furthermore, the adjustment method is more suitable for identifying changes in RH due to reduction in VPX, rather than to changes in temperatures.

At the end of the intervention study, tailored interviews were carried out in order to establish further the extent to which households had implemented the advice. Based on the interview results, each household was given an 'implementation score'. No correlation was found between the measured pre/post RH reduction and the 'implementation score'. Therefore, for those dwellings which experienced small reductions in RH, it is difficult to establish whether this was due to: a) lack of participants' action, b) adverse building characteristics hindering changes, c) limitations of the advice itself. However, during the interviews it also became apparent that participants experienced some difficulties in reporting their ventilation habits coherently.

5.0 The role of modelling techniques

The population model Mite Population Index (MPI)[2] was utilised in this study in order to: 1) Help identify those dwellings most at risk from mite growth; 2) Assess the effect of changes in hygrothermal conditions on mite populations. The MPI model predicts the likely effect of steady-state average hygrothermal conditions, on HDM population growth. The output is the MPI index, where for example 1.1 indicates 10% population growth and 0.9 indicates 10% population decline. The results (Table 3) obtained by utilizing measured pre-intervention average conditions indicate that the mite populations were rather stable during that period (average MPI $\cong$ 1), suggesting that even small hygrothermal changes could determine whether the population grows or declines (threshold effect). Dwelling 1 had the highest predicted daily population growth for both bedroom and bed. This is in agreement with the results from the dust samples. The other bedrooms with high MPI values did not have the highest allergen levels. This is

probably due to a reservoir effect, where for example the age of the mattress or cleaning regimes affect allergen levels.

Since the interventions included the removal of mites and their allergens, it was not possible to assess the *direct* effect of RH reductions on mite levels in the dwelling. Modelling was therefore utilised in order to assess the likely impact of hygrothermal changes on mite populations. In order to exclude the effect of changes in outdoor conditions, the monitored post-intervention temperatures and the *adjusted* post-intervention bedroom RHs were used as inputs in the MPI model. The use of adjusted hygrothermal conditions is theoretically equivalent to a situation where outdoor conditions stayed the same throughout the whole study, which allows the assessment of the likely impact of advice implementation on mite populations. Figure 3 shows a plot of the adjusted average hygrothermal conditions, with the pre and the post conditions joined by an arrow for each bedroom (numbers near data points correspond to the bedrooms' code). The plot includes a curve – corresponding to an MPI value of 1 – above which mite populations grow, and below which they decline. The results show that in all but Dwelling 8, conditions improved – independently from weather changes. Because of the threshold effect, even small reductions in RHs can lead to a reduction in mite populations (e.g. Bedroom 1 in Fig 3). Therefore, although the RH reductions obtained via changes in occupant behaviour may appear small, they could be sufficient in reducing mite infestations – particularly in winter times.

6.0 Discussion

This paper illustrated the methods and the findings of a pilot intervention study on HDM allergen avoidance, with a view to discussing their implications for a future larger scale study. The paper focused on the effects of tailored advice for heating and ventilation habits on household behaviour, indoor hygrothermal conditions and mite populations, as well as the role of modelling techniques. The results highlight the complexities associated with these types of studies, which are affected by several confounding factors[12]. Firstly, the study shows that, depending

on the time of the year, changes in outdoor conditions may result in improved hygrothermal conditions, thus confounding the effect of hygrothermal changes due to the advice implementation. Furthermore, occupant behaviour can change in relation to outdoor conditions. It is therefore important to adjust for changes in outdoor conditions in future studies.

Secondly, the study shows that changes in occupant behaviour can be difficult to assess – particularly with regard to ventilation habits. It is therefore vital that ventilation rates – not only air leakage – are measured in future studies, and a closer monitoring is carried out of occupant habits (for example through the use of participants diaries). Future studies might also need to focus on similar building types, in order to facilitate the assessment of changes in occupant behaviour. As demonstrated in this study, it is vital that *tailored* advice is provided, ensuring that the most effective intervention is carried out - since greater ventilation rates are not always desirable (e.g. leaky building). Changes in occupant behaviours can also be hindered by adverse dwelling characteristics. For example, in this study Household 1 was advised to increase ventilation rates, but their VPX did not decrease, most probably because of a combination of existing habits and their very airtight dwelling.

A further difficulty highlighted by this study was assessing the impact of changes in hygrothermal conditions on mite populations (and, indirectly, on health). This is because psychrometric control does not immediately affect HDM allergen reservoirs, which have to be removed in order to obtain any health improvement. However, allergen removal also results in killing the existing mite population, which therefore cannot be monitored for subsequent reductions due to hygrothermal changes. In any case, live mites are notoriously difficult to sample and mite populations would also be affected by changes in outdoor conditions. On the other hand, high allergen levels cannot be taken as a marker of favourable hygrothermal conditions, since a reservoir effect can be observed, for example due to the age of the substrate (e.g. mattress) and to cleaning regimes. Therefore, in future studies the use of population modelling techniques such as those utilised in

this pilot are recommended as a very useful tool for assessing the likely impact of hygrothermal changes on mite populations. Although the current models are based on steady-state data, transient models for mite microclimates are being developed by the authors, which will allow for more accurate predictions of mite populations. Furthermore, the authors have also developed an innovative technique for monitoring the impact of a dwelling's hygrothermal conditions on mite populations - described in a future paper. The development of models predicting the effect of hygrothermal conditions on allergen levels (as opposed to mite population only) is also recommended, since this would allow for a better understanding of the impacts of hygrothermal changes on respiratory health.

Due to practical constraints, the placebo effect and the role of other confounding health variables (e.g. sensitivity to other allergens) could not be adequately controlled for in this study and they should be properly addressed at the design stage in future studies (e.g. eliminating children who are allergic to other allergens). In this pilot one control dwelling achieved a large reduction in bedroom RHs – despite not receiving the advice until after the end of the study. This may have been due to the nature of this pilot (role of TV crew) but larger future studies should attempt to control for this by ensuring minimum interaction between participants, and with the project team.

This study showed that there was a statistically significant ($p<0.01$) decrease of measured bedroom RHs, which remained statistically significant although smaller, once the effect of changes in outdoor conditions was taken into account. The population modeling results indicated that during the pre-intervention period the mite populations were rather stable (average MPI $\cong$ 1), suggesting that even *small* hygrothermal changes could determine whether mite populations grow or decline.

7.0 Conclusions

This study suggests that in temperate climates even small changes in hygrothermal conditions can be crucial for the reduction of house dust mite infestations, particularly in winter. Tailored advice on heating and ventilation habits can lead to valuable changes in hygrothermal conditions. In some cases, however, these improvements may be hindered by occupants' reluctance to change and/or by pre-existing adverse building conditions.

The following recommendations are made for similar future larger scale studies:

- Ventilation rates and air infiltration should both be measured.
- An adequate method for taking account of changes in weather conditions should be adopted.
- Occupant behaviour should be monitored, for example by utilising relevant diaries and/or measuring the frequency of window and fan usage.
- Selecting the same building types might facilitate assessing changes in occupant behaviours.
- Allergen and mite samples are invaluable, and the results should be expressed in terms of both allergen concentrations and total allergen content/load. Personal air samplers might also be useful.
- Hygrothermal and HDM population models are very useful to assess the likely effect of hygrothermal changes on mite infestations. Ideally, these models should be able to predict the effect of transient conditions.
- The placebo effect, the role of other allergens and the effectiveness of controls should be adequately addressed at the study design phase.

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References

1 National Academy of Sciences. Clearing the Air. Washington: National Academy Press, 2000.

2 Crowther D, Wilkinson T, Biddulph P, Oreszczyn T, Pretlove S, Ridley I. A simple model for predicting the effect of hygrothermal conditions on populations of house dust mite Dermatophagoides pteronyssinus (Acari: Pyroglyphidae). Experimental and Applied Acarology 2006; 39: 127-148.

3 Cunningham MJ. Controlling Dust Mites Psychrometrically - a Review for Building Scientists and Engineers. Indoor Air 1996; 6: 249-258.

4 Arlian LG, Neal JS, Vyszenski-Moher DL. Fluctuating Hydrating and Dehydrating Relative Humidities Effects on the Life Cycle of *Dermatophagoides farinae* (Acari: Pyroglyphidae). Journal of Medical Entomology 1999; 36(4): 457-461.

5 Pike AJ, Cunningham MJ, Lester PJ. Development of Dermatophagoides pteronyssinus (Acari: Pyroglyphidae) at constant and simultaneously fluctuating temperature and humidity conditions. Journal Of Medical Entomology 2005; 42(3): 266-269.

6 Luczynska C, Sterne J, Bond J, Azima H, Burney P. Indoor factors associated with concentrations of house dust mite allergen, Der p 1, in a random sample of houses in Norwich, UK. Clinical and Experimental Allergy 1998; 28(10): 1201-9.

7 Coates D (Twenty Twenty Television), Producer of: How to beat your kid's asthma. Screened on Channel 4 on the 6 and 10 April 2006.

10 Platts-Mills TAE, de Weck AL. Dust mite allergens and asthma - a worldwide problem. Journal of Allergy and Clinical Immunology 1989; 83: 416–27.

8 Sherman M. The use of Blower Door Data. Lawrence Berkeley Lab Report 35173. Berkeley: Lawrence Berkeley Laboratory, 1998.

9 Oreszczyn T, Pretlove SEC. Condensation Targeter II: Modelling surface relative humidity to predict mould growth in dwellings. Building Services Engineering Research & Technology 1999; 20(3): 143-153.

10 Arlian LG, Veselica MM. Effect of temperature on the equilibrium body water mass in the mite *Dermatophagoides farinae.* Physiology Zoology 1981; 54(4): 393-399.

11 Sanders C ed. IEA Annex 24. Heat, Air and Moisture transfer through new and retrofitted insulated envelope parts. Final Report. Vol. 2, Task 2 Environmental conditions. Belgium: Laboratorium Bouwfysica, 1996.

12 Howieson SG. Housing and asthma. Oxford: Taylor and Francis Ltd. Spon Press, 2005.

Table 1 Baseline measurements for building characteristics, hygrothermal conditions and mite infestation levels.

Dwelling	[θ]Moisture Product. (kg/day)	Volume (m^3)	[Δ]Air Infiltr. (ach^{-1})	*Mites (mites/ m^2)	*Der p1 Conc. (µg/g)	*Der p1 Load (µg/m^2)	#Pre, VPX (kPa)	#Pre, % Time CEH>RH (%)	#Pre, Temp (°C)	#Pre, RH (%)
1	7.2	163.1	0.2	20.3	23.0	1.16	0.6	92.1	20.9	68.7
2	4.2	198.6	0.4	0.0	1.7	0.10	0.2	18.1	20.7	52.3
3	3.4	127.2	0.5	0.0	0.3	0.13	0.4	66.8	20.8	57.3
5	6.5	484.5	0.9	0.0	0.4	0.01	0.2	0.0	20.9	40.4
6[c]	11.9	286.2	1.1	21.7	0.3	0.11	0.3	36.3	20.6	54.1
7	13.7	189.8	0.6	17.7	21.4	2.30	0.2	73.4	18.7	57.5
8[c]	6.4	137.4	0.5	0.0	3.3	0.24	0.4	37.4	22.3	55.8
9	7.9	215.9	1.4	0.0	(-)	0.02	0.2	47.2	20.2	54.5
10	6.3	141.3	0.6	0.0	0.9	0.11	0.4	20.2	22.1	54.6
11	10.1	396.1	1.3	5.3	1.8	0.31	0.2	99.2	17.4	61.8
12a	5.3	263.0	0.6	1.3	2.2	0.26	0.3	90.3	(17.6)	57.4
12b	5.3	263.0	0.6	0.0	0.8	0.12	0.3	100.0	(17.4)	60.5
Average	**7.7**	**227.3**	**0.7**	**5.1**	**1.4[α]**	**0.16[α]**	**0.3**	**56.8**	**20.5**	**55.8**
Outdoor Conditions									**11.4**	**76.8**

[c]Control Dwelling; *Bedroom, Average of: Mattress, Floor, Pillow; #Child Bedroom; [θ]Whole dwelling, estimated; [α]Geometric Mean; [Δ](Air-infiltration measured at 50 Pa)/20; (-) Missing data. Note: central heating in Dwelling 12 was malfunctioning in the pre-intervention study.

Table 2 Post-intervention hygrothermal results, and difference with pre-intervention conditions, for the child's bedroom.

Bedroom Number	Post: VPX (kPa)	Pre-Post# VPX (kPa)	Post: Temp. (°C)	Pre-Post# Temp. (°C)	Post: RH (%)	Pre-Post # RH (%)	Post: % Time CEH>RH	Pre-Post# % Time CEH>RH (%)
1	0.6	0.0	19.4	1.5	59.8	8.9	76.3	15.8
2	0.1	0.1	19.5	1.2	40.7	11.6	0.0	18.1
3	0.2	0.2	19.6	1.2	41.9	15.4	0.0	66.8
5	0.0	0.2	19.2	1.7	37.6	2.8	0.0	0.0
6	0.1	0.2	18.0	2.6	41.6	12.5	1.8	34.5
7	0.2	0.0	17.4	1.3	47.4	10.1	9.5	63.8
8	0.5	-0.1	21.9	0.4	48.4	7.4	0.1	37.3
9	0.2	0	21.3	-1.1	38.4	16.1	0.0	47.2
10	0.3	0.1	20.4	1.7	43.5	11.1	1.2	19.0
11	0.2	0	17.8	-0.4	47.8	14.0	12.1	87.1
12a	0.0	0.3	17.3	0.3	40.3	17.1	5.6	84.7
12b	0.2	0.1	17.2	0.2	47.1	13.4	2.8	97.2
Average*	**0.2**	**0.1**	**18.9**	**0.8**	**44.5**	**12.1**	**10.8**	**50.0**
Outdoor	**(n.a.)**	**(n.a.)**	**6.6**	**4.8**	**80.2**	**-3.4**	**(n.a.)**	**(n.a.)**

#Pre-Post Difference; *Excluding controls (bedroom 6 and 8)

Table 3 Pre-intervention study: predicted daily mite-growth risk (daily MPI)

Bedroom	Daily Bedroom MPI
1	1.03
2	0.97
3	0.98
4	0.94
4	0.97
5	1.00
6	0.95
7	0.98
8	0.95
9	0.97
10	0.98
11	1.00
12a	1.00
12b	0.99
Average	**0.98**

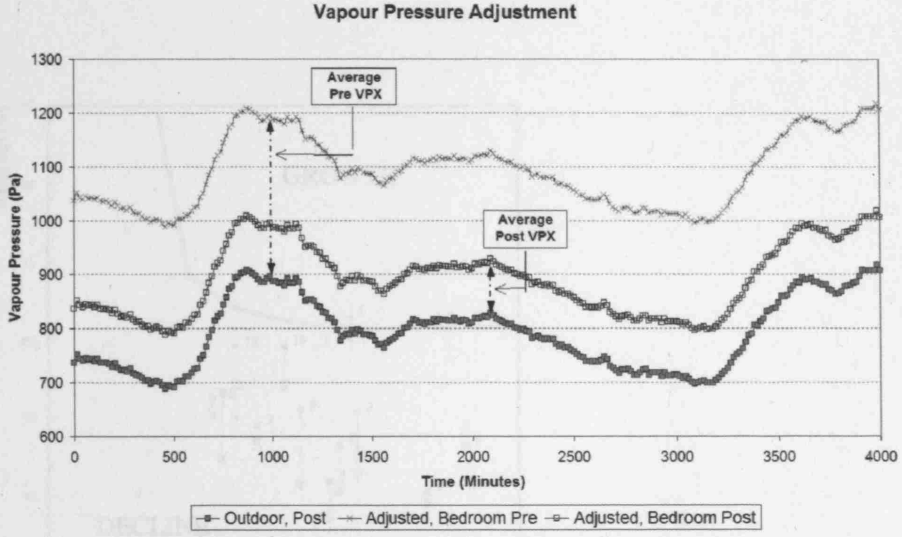

Figure 1: Schematic representation of the adjustment calculation for the vapour pressures, pre and post intervention.

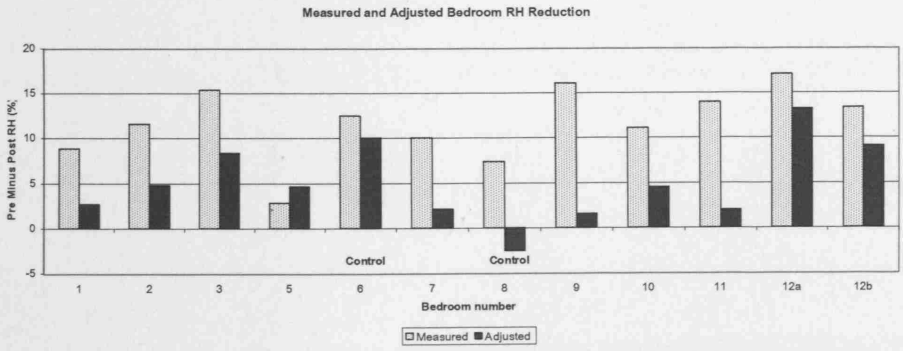

Figure 2: Measured and adjusted reduction in bedroom RHs.

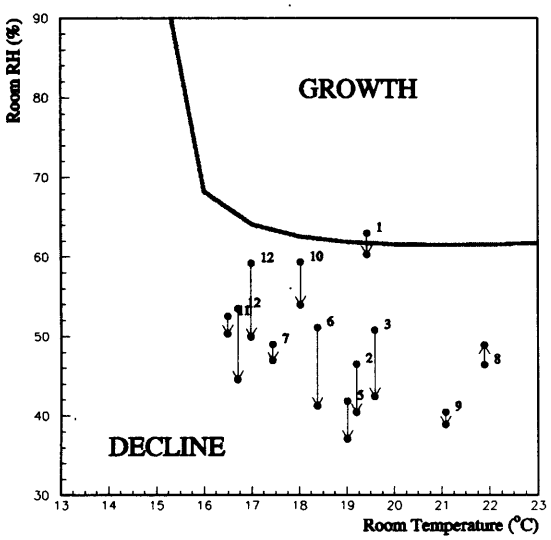

Figure 3: Predicted bedroom mite growth risk, using adjusted hygrothermal conditions: pre versus post intervention. The solid curve represents conditions where HDM populations are stable (MPI=1).